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WORKS OF E.J. BUTLER

FOUNDER DIRECTOR

COMMONWEALTH MYCOLOGICAL INSTITUTE

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THE IMPERIAL BUREAU OF MYCOLOGY

BY

E. J. BUTLER, C.I.E., D.Sc., M.B., F.L.S.

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THE IMPERIAL BUREAU OF MYCOLOGY.

By E. J. Butler, C.I.E., D.Sc., M.B., F.L.S.

The movement which led to the establishment of the Imperial Bureau of Mycology originated from several different sources.

In the first place it had long been felt by many that the remarkable success that had attended the formation of the Imperial Bureau of Entomology, in encouraging and co-ordinating entomological work in relation to agriculture throughout the Empire was justification for the establishment of a similar organization to deal with the other great class of destructive agencies in agriculture, namely the diseases of plants caused by fungi. Then the shortage of supplies during the war emphasized the importance of conserving raw products obtained from the vegetable kingdom, for the provision of which this country is so dependent on the agricultural prosperity of the Overseas Empire. The bearing of economic mycology, especially plant pathology, in this matter was brought into prominence by a discussion at the Newcastle meeting of the British Association in 1916, at which specific proposals were made for the establishment of an Imperial Bureau of Mycology as a co-ordinating and stimulating agency for helping on the study of the diseases of plants throughout the Empire. At the same time some of the mycological workers in the overseas parts of the Empire began to press for improved facilities in England to replace the help in certain directions which they had been accustomed to obtain from foreign countries, some of which became enemy countries in 1914.

Thus the movement from within met with a similar movement from outside. The proposal to establish a Bureau of Mycology in London was laid before the Imperial War Conference in 1918, and, backed by the support of that powerful body, was referred to the Overseas Governments for consideration. The latter approved, and undertook to finance the Bureau for a period of three years in the first instance. On the initiative of the Colonial Office a Managing Committee was formed, comprising a number of well-known biologists with Viscount Harcourt as their Chairman, and the Bureau commenced work towards the end of last year. Quarters have been found for it in the former laboratory of the Plant Pests Branch of the Ministry of Agriculture at 17, Kew Green, as this Branch has been transferred to Harpenden. At Kew the Bureau has the advantage of proximity to the fine library and collections of the Royal

Botanic Gardens and it will work in close co-operation with the latter institution.

The following is a brief sketch of the proposed activities of the Bureau, which have been worked out in consultation with mycologists both here and in the colonies. As its primary function is to be of use to economic mycologists in the overseas parts of the Empire, the views of the latter have naturally great weight, and it is already evident that a great deal of consideration is being given by the Colonial Departments of Agriculture to the best methods of making the Bureau of real use. After having had nearly twenty years practical experience in India of the directions in which help is needed it was not difficult for me to suggest what the broad lines of our work should be; other useful proposals have been sent in from abroad, and there is no doubt that still more will follow.

There appear to be two main directions which must be followed if the Bureau is to justify its existence; and there are many subsidiary activities which will greatly add to its utility.

The two main directions are of equal importance. They are the accumulation and distribution of information on all matters connected with the diseases of plants, and the identification of specimens of injurious fungi.

For the former the Bureau must endeavour to keep in touch with mycological and pathological work going on in all parts of the world, and must index all the useful literature so that past and current work on any particular subject can be readily traced. The oversea worker is often in too isolated a position to be able to do this for himself: his first need is to know what has been done elsewhere on the problems with which he is faced and whether there is any recent or current work on them; and for this we hope to provide the necessary organization at the Bureau. The number of journals, periodical reports and the like that will have to be examined is very large, probably approaching four figures, but many of these have only an occasional reference of value. As soon as the necessary additional funds are provided (the original proposals in 1918 were calculated on a war basis) it is proposed to start a periodical abstracting journal on the lines of *The Review of Applied Entomology*, published by the Imperial Bureau of Entomology. But it is clear that this in itself will not be enough. We must be able to issue, on demand, the complete references to past work on the problems on which individual workers overseas are engaged. It will naturally take time before we can build up complete subject catalogues to enable this to be done readily. Besides the general subject catalogue we will require to compile, as soon as possible, a host-plant index, giving the fungi recorded on economically

important plants, for which the basis already exists in Vol. XIII and the subsequent volumes of Saccardo's *Sylloge Fungorum*, but which requires to be amplified in the direction followed in Oudemans' *Enumeratio Systematica Fungorum*. The latter only deals with the European flora, includes all plants, economic or not, and is of very limited value to, say, a worker in the tropics, as will be readily realized when we find only a single entry under the family *Zingiberaceae*, which includes such important crops as ginger and turmeric, both subject to several serious diseases. In addition to supplying references we must be prepared also to furnish abstracts and, in certain cases, to lend out original papers. This will be of great value to isolated workers and I attach great importance to it.

The next main line of work is to organize a system for the prompt identification of injurious fungi. In this respect mycologists have not at present as good facilities as those possessed by entomologists. Specialists in particular groups are not as readily available to us as to them; we are fewer in number and the amateur who specializes in a particular group is greatly to seek when once we descend below the higher fungi and enter the groups to which the majority of the injurious species belong. The organization here will have to be to some extent international as it is in entomology, and we must aim at being able to refer a species in a particular family or genus to the recognized authority on that group—when there is one. If, as I hope and expect, very large collections of fungi from the tropics and elsewhere are sent in, the Bureau can provide such material as to make it very attractive to systematic mycologists to specialize in certain families.

Arising out of this branch of the work is the comparative study of pathogenic fungi. This should be an important feature of the work of the Bureau and there is urgent need for the critical examination, with culture work, of such tropical pathogens as the *Phytophthoras*, *Diplodias*, *Glomerellas*, etc. When this work is under way there is no reason why it should not be developed into a culture-supplying organization, primarily to deal with tropical and sub-tropical forms. The chief difficulty in this is the want of suitable culture rooms at the Bureau, but a recent very helpful offer from the Lister Institute has suggested a way out.

Gradually there should be at the Bureau a very good herbarium of parasitic fungi. Such a collection will be essential for our own systematic work—for when all is said and done there will be a great deal of systematic work which the Bureau will have to undertake itself—and will be invaluable for reference purposes. Duplicates will be kept in as large quantities as

possible and named collections issued to various centres, especially the laboratories overseas. Types will not be kept at the Bureau as the collections there will be essentially working collections, the types being preserved at the Kew Gardens Herbarium or, in the case of specimens dealt with by outside specialists, at some centre agreed on in advance.

So far the Bureau has been considered as a centre for accumulating information and a clearing house for referring such information to the proper source, and as a centre for the systematic and cultural study of injurious fungi and an agency for getting the parasitic fungus flora of the overseas parts of the Empire worked out.

Nothing has been said about the prosecution of original research into the diseases of plants. The diseases with which we are concerned are those occurring in the distant parts of the Empire, and I hold strongly the view that true pathological research can only be done on the spot, where living clinical material is available. But I hope that the Bureau will become a place to which the overseas worker will naturally gravitate when in England on leave or otherwise, and every effort will be made to provide him with well-equipped laboratories where he can continue the study of problems in which he is interested, in directions which can be done at a distance from the crop concerned, and which can be checked and controlled by his own first-hand knowledge of the local conditions. I hope research work of various kinds will go on at the Bureau, but I hope equally that it will never be believed that the Bureau can replace the local worker, for that is not true.

Most countries now legislate for the exclusion or control of injurious fungi. Each country has usually worked out its own methods of dealing with this question and there is need for a central clearing house which can supply authentic information regarding the practice elsewhere. I am collecting the various acts and ordinances and I hope we will be able to notify changes or give other useful assistance in connexion with this matter.

It is often difficult for the isolated worker to procure the most suitable spraying or other appliances or to keep entirely up-to-date in the practical methods of dealing with fungus diseases. Here again I think we can usefully help by keeping in touch with manufacturers and with those who are testing new methods.

Apart from the diseases of plants, there are two other branches of applied mycology to which a brief allusion may be made, the study of the diseases of animals and man caused by fungi and the study of the fungi that are of importance in technical manufactures and trades. There is a vast field in our tropical

countries as yet almost unexplored (if we except the work of Castellani) in regard to the former of these subjects and I hope we can do something to stimulate research in it, and perhaps help in its more purely mycological side. Whether the Bureau can take any active part in the study of technically important fungi or not it is as yet difficult to say, but I have already had enquiries of this nature both in India and since I joined the Bureau, and I think there is a need in this direction also which we might do something to fill.

To sum up, it seems to me that the Bureau can be most usefully engaged if it has amongst its functions:

(1) The accumulation and distribution of information on all matters concerning the diseases of plants caused by fungi, of interest to workers in the overseas parts of the Empire.

(2) The publication of a periodical abstracting journal on the lines of *The Review of Applied Entomology*.

(3) The formation of a good reference library, especially of original papers in the reprint or "separate" form which will be available for issue on loan.

(4) The organization of a system for securing reasonably prompt identification of injurious fungi.

(5) The critical study of parasitic fungi which are at present the subject of confusion, such study to be in large part based on cultural methods; and the arrangement of facilities for the supply of authentically named cultures to overseas workers.

(6) The formation of a good working herbarium of parasitic fungi, with larger specimens and photographs illustrating diseases not known in this country; and the supply of named collections to other institutes and individuals.

(7) The provision of laboratory facilities to enable overseas workers to continue work, in problems in which they are interested, when in England.

(8) The organization of a central agency from which up-to-date information on such matters as legislation against plant diseases caused by fungi and recent progress in methods of control by spraying and the like can be supplied.

(9) The stimulation, as opportunity arises, of the study of other branches of applied mycology, such as those referred to above as being of interest in medicine and technology.

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Some Relations between Vegetable and Human Pathology.

BY

E. J. BUTLER, C.I.E., M.B.

Director of the Imperial Bureau of Mycology.

(Paper read at a Meeting of the Royal Society of Tropical Medicine and Hygiene, Thursday, 19th January, 1922.)

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SOME RELATIONS BETWEEN VEGETABLE AND HUMAN PATHOLOGY.

BY

E. J. BUTLER, C.I.E., M.B., *Director of the Imperial Bureau of Mycology.*

(Paper read at a Meeting of the Royal Society of Tropical Medicine and Hygiene, Thursday, 19th January, 1922.)

The differences in the organisation of the higher plants and animals are such—especially in the absence of true circulatory, nervous and digestive systems in plants—as to preclude any comparison between the great mass of the disorders from which the two kingdoms suffer. But since, in their ultimate structure, they are composed of the same cellular units, and since they are exposed to the attacks of similar parasites, it would be strange if there were no points of contact between vegetable and animal pathologists.

Vegetable pathology is a younger science than that of man and the higher animals, and there are, as yet, no handbooks that give in any detail a comparative survey of the pathological processes in plants.

But, I think, there are grounds for hoping that the older science may yet gain something from the study of certain disease phenomena in plants. The directions in which this may be sought are in certain branches of cellular pathology; in the diseases involving changes in physiological processes, such as altered metabolism; in certain classes of parasitic and virus diseases; and in the effect of environmental conditions on the interaction of host and parasite.

I propose to give only a very few instances of disease processes in plants that have some analogy to those in the animal kingdom, but without attempting to define their exact bearing on particular aspects of human pathology. While they are not specially confined to the tropics they are prevalent there, and my own actual experience has been almost entirely tropical.

The first is tumour formation in plants, as exhibited by the well-known crown gall tumour caused by *Bacterium tumefaciens*. I have recently had an opportunity of going over with Dr. Erwin F. Smith, of Washington, the work on this disease on which he has been engaged for the last eighteen years. It is regarded by plant pathologists, the world over, as one of the masterpieces of scientific work in their field, and has raised Dr. Smith to the position of a recognised leader amongst them.

The tumours produced by this organism on a multitude of plants are usually hyperplasia caused by the repeated division of the cells to produce a solid small-celled tumour tissue without cavities. The cells are often markedly embryonic in character, and divide mitotically and amitotically. Secondary tumours frequently develop, often at a considerable distance and deep-seated in the tissues. They are not caused by migration of the bacteria across normal tissues, but by a definite outgrowth from the primary tumour in the form of a tumour-strand of infected cells of marked characters, and easily recognised in section, though often only a few cells thick. Such strands have been traced for ten inches in length. The secondary tumours have the characters of the primary, so that if a leaf bears tumours from one on the stem, their structure will be a stem structure. Secondary tumours can also be produced by grafting a part of a primary tumour on a suitable part of the plant. Growth of the tumour is unlimited and devoid of polarity, and the destructive action on the plant is usually merely the result of pressure and crushing, though necrosis permitting secondary infection is common.

The structure of the tumour varies according to the tissue primarily infected. Most of them are of the ordinary ground or conjunctive tissue cells, *e.g.*, in the cortex or pith, and are the nearest approach that one could expect to get in plants to Sarcomata. There is no epithelium in plants, and the epidermal tissues are thin, usually a single layer, and hard to inoculate without involving the underlying ground tissue. But a few cases have been described in which the tumour appears to be composed mainly of epidermal elements, including hairs (the plant hair being an outgrowth from a single epidermal cell), and these may be compared with Epitheliomata. The most interesting type, however, is Embryomata. These were first detected as recently as 1916. They are composed of a jumbled mass of young shoots and roots of usually incomplete structure, having only bits of organs, but sometimes forming more or less complete dwarf shoots. The secondary tumours developed from these have usually the same embryonic teratoma structure.

The organism which causes crown gall is a cell parasite of a highly developed type, in which the host cell is not destroyed but is stimulated to increased activity. There are many such cases in plant parasites. To the best of its ability the parasite aims at preserving the life of the host cell for its own needs. That it over-stimulates the cells, causing death by crushing and necrosis, may be regarded, more or less, as an accident.

Bacterium tumefaciens can be isolated and cultivated so that its morphology and characters are well known. It is rare in the tissues, at least in an active state; in one case it was estimated that there were only 200 living bacteria per cubic centimetre of the tumour. But perhaps the most significant thing that I learned from Dr. Smith was that, contrary to his earlier belief, he is not now certain that he has ever recognised the organism in the tissue. What he previously saw he now thinks were only cell inclusions or chondriomes. Thus we have a parasite capable of causing a tumour having all the characters of a malignant growth; the organism has been isolated, grown in pure culture, and new tumours produced at will with it, but it is not with certainty to be recognised in the tissues of the tumour.

More recently Dr. Smith has been able to produce experimentally small tumours of limited growth, having the characters of incipient crown galls, by injecting in suitable parts of plants certain of the diffusible products of the metabolism of *Bacterium tumefaciens*, including ammonia, amines,

organic acids, etc., and he considers that the tumour is ordinarily caused by the continual production of small quantities of these excreted products by the parasite.

I am aware that the path of cancer research is strewn with the wrecks of parasitic theories; but the recent work on plant cancers, the experimental production of embryonic teratomata, the extreme difficulty of detecting the parasite in the cells (it is certainly *in*, not between the cells), and the evidence that it acts by the production of repeated small quantities of an excreted stimulus, makes it easy to understand the view held by Dr. SMITH, and other vegetable pathologists, that cancer will ultimately be found to be due to some similar cause.

A second direction in which disease phenomena in plants may throw light on certain animal diseases is found amongst the obscure group of diseases which the vegetable pathologists are calling "virus" or "contagium" diseases. These are diseases in which etiological studies suggest the presence of parasitic agent, but in which, with the means at our disposal, no parasite can be detected, and filtration through the Berkefeld filter does not cause a reduction in virulence.

The number of such diseases in plants is large, but many of them have only recently been recognised. They have all certain characters in common, but they fall into several groups, the most important of which is that known as the "mosaic" group, because its most obvious symptom is a variegated appearance of light and dark green patches on the leaves. So rapidly have new cases of mosaic been recorded in the last few years that one is tempted to say that every cultivated plant has at least one such disease. Cross-inoculations show that while allied plants (as, for instance, potato and tomato) may suffer from the same mosaic, the mosaics of distantly related plants are usually distinct.

These diseases are constitutional, the virus reaching all parts of the plant in three to four days after inoculation in the mosaic of cucumber. The incubation period before the first symptoms appear in this mosaic varies from four to fifteen days, and in potato and cabbage mosaic from twenty to thirty days. In sugar-cane mosaic the virus is restricted in its distribution, and may not reach every shoot. Many tissues and organs, though containing the virus, do not react to it in any way that has been hitherto detected. Also "carriers" have been found, *i.e.*, plants that show no symptoms after inoculation, yet contain infective virus in a

transmissible form. Actual tissue reactions (cell modifications) are often caused in the leaves and fruit. Necrosis of the light areas in the leaves also occurs not uncommonly in the older stages. The other symptoms are due to deep-seated alterations in the physiological activities of cells and organs, revealing themselves in altered metabolism and abnormalities of growth (dwarfing, crinkling or folding of flat surfaces, shortening of axes, and, at times, proliferation). The disease is not usually fatal in annual plants, though death from weakness may occur. The reduction in vigour may be very great or slight. Mosaic and allied diseases of potato in England may cause a reduction of over 50 per cent. in the yield, and in Bermuda losses up to 75 per cent. have been recorded. On the other hand, I recently saw fields of sugar-cane in Louisiana with 100 per cent. infection from mosaic, but was informed by the local plant pathologist, EDGERTON, that the loss of yield was slight, though the disease has been present for seven years, and in other localities, *e.g.*, Hawaii, has been known to reduce the sugar yield by 60 per cent. The so-called deterioration of many cultivated plants, such as potatoes, leading to the constant demand for new varieties, has recently been recognised to be, in many cases, the result of the gradual accumulation of some one or other of these virus diseases.

From the standpoint of comparative pathology, what is important is the knowledge that has already been gained as to the methods of transmission and certain other characteristics of these virus diseases.

So far as is known all these diseases are insect borne. There are other methods by which infection can be produced, but the normal method in nature is by insects, chiefly sucking insects, especially aphids. In no case has transmission by mere contact been demonstrated. In one case, curly top of beet, no other method of infection than through the insect (a leaf hopper, *Eutettix*) has been discovered, and in this case the insect has to be kept for a period of twenty-four to forty-eight hours after feeding on a diseased plant before it can inoculate a healthy one, after which it can remain infective for 100 days. This suggests that a part of the life-cycle of the parasite is passed in the insect. In most cases, however, the virus can be obtained by extracting the juice (cell sap) of a diseased plant, and can be successfully inoculated on a healthy plant by injection into any part of the plant (tobacco mosaic) or into certain parts only (growing point in sugar-cane mosaic, any part but the root or flower

in cucumber). It can also be transmitted often by crushing a diseased leaf on a healthy one, so as to injure both tissues, or even (cucumber mosaic) by bringing the broken hairs of healthy and diseased leaves into contact. In such cases the virus is evidently in each individual cell. In spinach blight the crushed bodies of aphids that have fed on diseased plants are infective. In this disease, too, the offspring of infected aphids are infective occasionally. In certain cases the disease is carried in the seed borne by diseased plants, but in a good many this does not occur. There is much evidence that the virus travels in the plant through that part of the vascular system, the phloem, in which the elaborated sap is conveyed. This system does not reach to the seed, the plant embryo feeding entirely by osmosis. It is most interesting to note that recent studies show that certain aphids, including some that are known to carry mosaic, feed by inserting their very fine and flexible probosces into the cells of the phloem, avoiding other cells as a rule.

One other method of transmission must be mentioned. Even in the most refractory cases, as beet curly top, infection can be secured by grafting a diseased on to a healthy plant, provided organic union is obtained.

After extraction from the plant, the life of the virus varies from only twenty-four hours (sometimes in cucumber mosaic) to a year or more (tobacco mosaic if dried). It is killed by a temperature usually of between 70°C. and 80°C., but can withstand exposure to very low temperatures. In some cases the ordinary antiseptics kill it, but the tobacco mosaic is resistant to most weak antiseptics. The concentration in the sap is such that a needle drop can be diluted to 1,000 without any loss of virulence, though virulence is reduced by a dilution to 10,000 (tobacco and cucumber). Filtration through porous filters depends on the size of the pore. The cucumber mosaic virus filters through the Berkefeld but not through the Chamberland; tobacco mosaic filters through both, but is retained by powdered talc.

Though there are still a few vegetable pathologists who prefer the enzymic or toxic hypotheses to the parasitic explanation of these diseases, most agree that their study has made it necessary to assume that they are caused by ultramicroscopic living organisms. The mere fact that they have not been successfully grown *in vitro* has no weight with the vegetable pathologist who is familiar with whole groups of parasitic fungi,

such as the rusts, not one of which has ever been grown apart from its host plant. It is as certain as anything can be that no visible organism is present in extracted cell sap that is known to be highly infective. This does not preclude the possibility that the organism may have a visible stage, and indeed quite recently two independent workers have claimed to have discovered, in cells of sugar-cane and maize suffering from mosaic, bodies which in one case may perhaps be suggestive of a parasite not unlike a stage in *Rickettsia*.

Another set of problems, of no less interest to the vegetable than to the animal pathologist, is the geographical and seasonal distribution of certain diseases.

The experimental study of the relations of plants to their environment is naturally much easier than with human beings. In our botanic gardens we habitually grow tropical plants with success, being able to imitate their natural conditions. It is only recently, however, that plant pathologists have made use of their opportunities to study the influences of such climatic factors as humidity and temperature on plant disease. The results have been remarkable. It has been found possible to correlate the geographical distribution of a considerable group of plant diseases with the temperature and humidity reactions of the parasite and its host.

In the simplest cases a single factor, such as the degree of relative humidity of the air, acts by limiting the development of the parasite. One of the best examples of this is a disease of the rice plant which I studied in India. It was strictly confined to a large rice area at the head of the Bay of Bengal, and while the main crop from July to December was often totally destroyed, the secondary crop in the spring entirely escaped, though it was shown by artificial inoculations to be equally susceptible. I found that the organism could neither move nor feed nor reproduce at an air humidity of below 90°, while, though it is actively mobile, it is unable to feed or reproduce in water. To be able to multiply and attack the rice plant it therefore requires moderately prolonged periods when the air humidity is over 90°. This it only gets in certain limited areas in India during the second half of the year; and at a time when the disease was causing considerable alarm, it was possible to state that the bulk of the rice-growing area was not threatened.

The meteorologic factor may act by affecting the receptivity of the host-plant. Thus in the vine mildew, infection is dependent on the

condition of the stomata, through which the parasite enters. If these remain semi-closed, as in young leaves or in dry conditions, infection is impossible. Infection has been found to fail if the moisture of the soil in which the vine is growing is below 15 per cent., unless the air humidity is above 80 per cent. If the soil moisture is above 20 per cent., the stomata open widely enough to permit infection at air humidities above 40 per cent.

But it would seem that in many cases the action of the meteorological factors on both the parasite and the host in their mutual relations—on what may be, for convenience, termed the host-parasite complex—has to be taken into account. In the onion smut, for instance, it is possible to get 100 per cent., or no successful infections, according to the soil and air temperatures used. The onion seed will germinate at soil temperatures from 10°C. to 30°C. but the best top growth is got with the soil temperature of 20°C. to 25°C. Infection by the smut fungus can be got freely from 10°C. to 25°C. but is slight at 27°C. At a soil and air temperature of 25°C. a great many plants will outgrow the infection and recover, but at an air temperature even as high as 30°C. to 33°C., infection can be got if the soil temperature is kept below 25°C. In outdoor plantings the disease was completely inhibited when the mean soil temperature reached 29°C., and increased progressively with lower temperatures. The relation is believed to act chiefly through the host-plant, as successful incipient infection can be got at the higher temperatures, but the subsequent development of the fungus is inhibited. The geographical distribution of the disease in the United States, where it is absent from large areas in which onions are sown in the warm season, has been completely explained by this work.

Recently it has been shown that the severe outbreaks of epidemic malaria in the Punjab are governed by a set of temperature and humidity factors affecting both the malarial parasite and the insect host. With a warm-blooded animal like man the temperature factor is probably of slight importance, except in so far as it acts on the parasite while exposed to the air, as in the external air-passages. But in insect-borne diseases, and in the life of the parasite outside its host, these studies have a certain interest, and the recent discovery that so small a difference as 5 per cent. relative humidity or 3°C. or 4°C. temperature can cause a difference of 100 per cent. in infection has come as a surprise to many plant pathologists.

In these few instances, selected from widely different fields, I have tried to show that the problems which face the vegetable pathologist are often akin to those of human pathology. Where there is any evidence that disease processes are fundamentally alike in plants and animals, I venture to think that they are worth careful examination by pathologists generally. Their study in plants has many obvious advantages in the ease of experimentation and manipulation.

Vegetable pathology is under a great obligation to the older science, from which many of its methods and principles are borrowed, and there could be no more fitting way of repaying this debt than by the discovery that the study of plant diseases may throw light on some of the more obscure problems of human pathology.

ROYAL BOTANIC GARDENS, KEW.

BULLETIN

OF

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PHYTOPATHOLOGY IN THE UNITED STATES.

E. J. Butler.

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ROYAL BOTANIC GARDENS, KEW.

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No. 2] [1922

X.—PHYTOPATHOLOGY IN THE UNITED STATES.

E. J. Butler.

With the permission of the Committee of the Imperial Bureau of Mycology the Report by Dr. E. J. Butler, Director of the Bureau, on his visit to America during July and August 1921 is here reprinted with a few omissions and alterations. We are glad to have been allowed to bring the Bureau of Mycology and its value to the Empire to the notice of a wider public through the medium of the *Bulletin*.

The immediate purpose of my visit to the United States was to attend a Conference on the diseases of cereals held by the American Phytopathological Society at St. Paul (Minnesota) and

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Fargo (North Dakota) from July 19th to 23rd. The more important objects were, however, to visit a number of the chief centres of agricultural and botanical research in the eastern, southern and middle western States, and to meet as many of those engaged in research and the organization of research in these subjects as was possible in the time at my disposal. The specific aims that I had in view were : (1) to bring the international aspects of the work of the Imperial Bureau of Mycology before our American colleagues; (2) to enlist their co-operation in certain branches of this work, and to ascertain what assistance we might expect to obtain in the United States; (3) to examine the organization and present development of the study of plant pathology and its application to the prevention and control of crop diseases in the United States; and (4) to visit some of the chief crop areas of the eastern half of the country and see the conditions under which the crops are grown and the measures taken to improve varieties and methods. The time at my disposal was insufficient to carry out this last adequately.

The Conference on cereal diseases was attended by some fifty members of the American Phytopathological Society (including several Canadians), a few American visitors, and representatives from England, Japan, Australia, and the Philippines. It met at St. Paul from the 19th to the 21st of July, moved to Fargo on the night of the 21st, and terminated there at 10 p.m. on the 22nd. The daylight hours were mostly given to field meetings at the experimental farms at St. Paul and Fargo and automobile trips through the wheat belt of Minnesota and North Dakota, while meetings to discuss specific subjects were held in the evenings. The members were also taken through one of the largest elevators and flour mills in Minneapolis. The Conference concerned itself chiefly with the cereal rusts, particularly with the control of black rust by breeding resistant varieties, and the campaign for eradicating the barberry, on which this parasite spends part of its life; with the diseases caused by *Helminthosporium*, *Fusarium*, and *Sclerospora*; and with the newly introduced " take-all " and " flag-smut " of wheat. There is probably more work being done on these subjects than on any other branch of plant pathology in the United States at the present moment, and we had the benefit of having this work explained, and in some cases demonstrated, by those actually engaged in it. Educationally, the Conference was of considerable value.

Both before and after the Conference I visited a number of scientific institutions east of the Mississippi from Michigan to New Orleans, with a view to establishing direct relations with American mycologists and plant pathologists. Owing to the prominence given to the study of these subjects there are at present more men at work on them in the United States than in any other country. This work is being pursued not only in institutions specifically for applied science, such as the Bureau of Plant Industry at Washington and the State Experiment Stations,

but also in many of the Universities, such as (amongst those I visited) Cornell, Purdue, Illinois, St. Paul, and Wisconsin. The study of pure systematic mycology is, after a period of neglect, again showing marked evidence of vitality so that the lead taken by Germany in this branch will, I think, pass to America in the future. It seemed most important to enlist the interest of American workers in the Bureau of Mycology, and I found this the more easy in that the entomologists in several of the institutions visited were very familiar with the Imperial Bureau of Entomology in London and spoke highly of its value. At Wood's Hole, and the Cereal Conference, I met a number of members of the staff of institutions that I was unable to visit. Considerable interest was evinced in what was regarded as a new departure likely to be of value to workers in other countries. Most of the institutions visited are prepared to exchange their publications with those of the Bureau. In several cases I asked if I could count on the direct assistance of specialists in particular subjects, and in all these found the greatest readiness to co-operate. I am sure that our work will be supported and encouraged very generally in America, and that we can get an immense amount of help there. In Washington, in particular, I was promised invaluable assistance, everyone I approached, from Dr. Taylor, Chief of the Bureau of Plant Industry, downwards, being most cordial. The National Research Council and the Editorial Board of *Botanical Abstracts* were other bodies that undertook to give us any help they could, and the discussions I had with members of the latter Board were particularly useful in helping to determine the lines on which our new abstracting journal can be made most serviceable.

The trend of research work in America is towards specialization, as it is elsewhere, but American workers have been accused of carrying this to too great a length. This is, I think, to misunderstand the organization of research in that country. Though the individual is specialized, problems are approached from a wider angle than probably in any other country. One of the best illustrations of this can be got from the "project plan" of the organization of work in the Department of Agriculture. This is merely the co-operative investigation of a problem by a group of specialists under a project leader, each worker taking a particular aspect of the problem in which he is specially competent, but all keeping in touch with one another and with the project leader. The greatest care is taken, as a rule, to preserve the independence of the individual and to give the fullest play to original initiative and freedom of research. The inquiries are carried out at various centres, Federal and State officers co-operating in the work, and the former being posted, when necessary, to the State centres most suitable for the purpose. Half-a-dozen men may be working in several different States in close co-operation and under a common leader, but each carrying out a different side of the investigation, and with complete freedom of action.

The system was a result of the frank recognition of the fact that many, perhaps most, of the chief inquiries that needed to be taken up were beyond the capacity of any single individual to bring to a successful conclusion within a reasonable time, and that, for their solution, the separate aspects of each problem required very highly competent specialistic work, such as can only be acquired by deep or intensive study rather than by a broad survey. I believe this is the correct way to attack such problems, and that it is only in very rare cases that an investigator can be found whose knowledge of the several distinct aspects which the problem may present is deep enough to enable him to explore all or many of them adequately.

It is worthy of note that the National Research Council is working along somewhat similar lines. It is an organization established in 1916 by the National Academy of Sciences to advise and assist in applying the scientific and technical resources of the country to the objects of the War, and made permanent by an Executive Order of President Wilson's Government in 1918. It is not, however, in the usual sense a governmental institution. Its chief objects are to organize research, to stimulate research and its application, to formulate comprehensive projects of research and to promote co-operation " in order to secure concentration of effort, minimise duplication, and stimulate progress; but in all co-operative undertakings to give encouragement to individual initiative." It is controlled by its own members, who include representatives of the chief scientific and technical societies, government representatives, and members at large amongst whom, in 1920 21, were Mr. Elihu Root and Mr. Herbert Hoover, together with several leaders in industry, engineering, and the Press. It is supported by other than Government aid, but maintains close contact with Government departments in several of its divisions. In its attempts to bring together scattered work and workers, and to assist in co-ordinating scientific attack on large problems, it has largely adopted the " project plan," by the establishment of special committees of experts, which plan modes of attack and undertake to find men and means for carrying out the plan. There are about eighty such committees, and many of them have obtained appropriations from industrial and agricultural sources for specific research, *e.g.*, one from the Southern Pine Association of $10,000 for maintaining certain forestry researches, and one which is now being negotiated with a group of tobacco growers and manufacturers for the study of tobacco diseases. Through the Council, there is also being organized an Institute for Research in Tropical America which will have a very definite bearing on some aspects of the work of the Bureau of Mycology. I had the advantage of discussing these and other activities of the Council with Dr. L. R. Jones, Chairman, and Dr. McClung, past Chairman, of the Division of Biology and Agriculture, and Dr. Yerkes, Chairman of the

Research Information Service of the Council, who is Resident Director of the Service at the headquarters in Washington. The opportunity I had of examining the working of the latter service was of particular interest, since it is engaged on work very similar to the "informational" side of the work of the Bureau of Mycology.

The question of abstracting and indexing scientific literature is occupying the attention of various organizations in America. A joint committee of the National Research Council and the American Association for the Advancement of Science was formed to consider this matter in 1920, and the question of taking steps for the establishment of an international institute for scientific bibliographical work is being examined, Dr. Kellogg being now in Switzerland on behalf of the National Research Council with a view to reporting on the utilization of the Concilium Bibliographicum at Zurich as a nucleus for the proposed institute This is a matter of direct interest to the Bureau of Mycology.

In a general way, much more attention is being paid to the "business" side of the organization of scientific work in the United States than elsewhere, and this is coupled with the development of "team work" in attacking important problems. They are recent developments and have not yet begun to produce their full effects, but the thoroughness of the preliminary preparation and the numbers who have combined to give effect to these aims make it certain that the result will be a vast stimulation of research activities. It was several times remarked to me that the formation of the Bureaux of Entomology, Tropical Diseases, and Mycology in London was evidently in response to similar needs, and that we should keep in close touch with one another. In the development of closer international relations, the trend in America is in favour of separate national organizations in the different countries with close mutual relations rather than single international institutions such as the International Institute of Agriculture at Rome. In our work we cannot fail to develop international contacts, and the sympathetic attitude towards such contacts (especially with the British Empire) which I found to be pretty general in the States will be well worth consolidating. I found a very wide recognition of the truth that phytopathology is as definitely an international interest as public health, and in several addresses at functions which I attended the formation of the Bureau of Mycology was welcomed on account of the international significance of its work.

The chief places visited with the special object of seeing the work on crop improvement and diseases were St. Paul (Minnesota) for cereals, Fargo (North Dakota) for flax, Baton Rouge and Audubon Park (Louisiana) for sugarcane and rice, Hartsville (South Carolina) for cotton and maize, New Haven (Connecticut) for tobacco and maize, St. Catherines (Ontario, Canada) for fruit, Lansing (Michigan), Lafayette (Indiana), and Urbana (Illinois) for cereals, fruit, and vegetables, and Cornell (New York) for maize.

The principal objects were to see what work was actually being done in certain areas on these crops, and to get into personal touch with the men in charge of this work so as to facilitate future relations. The chief value of this will be that I shall be better able to apply or to refer applications from overseas correspondents of the Bureau to the most likely American source of assistance in special inquiries. Want of time prevented me from fully carrying out my programme in this direction. What I saw convinced me that America is far ahead of other countries in the general application of modern methods of plant breeding and the use of fungicides for the reduction of the wastage due to disease. Maize, wheat, cotton, beans, sugarcane, tobacco, and fruit were the principal crops seen in which these methods were being applied with the greatest success.

The very great attention directed to this matter in the United States is the result not only of the magnitude of their agricultural industry, but also of the generally great severity of the diseases of their crops. On the whole, the diseases of plants that have an economic value are distinctly worse in the United States than in Europe, though not worse than in some of the overseas parts of the British Empire. The reason for this appears to be that there are two factors concerned in intensifying diseases in countries that are newly opened up to settled agriculture. (1) the crops grown are largely exotic, and hence become exposed to the attack of indigenous parasites against which they have not developed powers of resistance; (2) with the introduction of new plants of economic value, exotic parasites are brought in, and some of these develop into serious pests of pre-existing plants in their new home. In Europe both these factors doubtless operated in the past, and the latter was responsible for the enormous ravages of introduced vine diseases in the last century. But the process has been very gradual in Europe as compared with America, and the centuries-long history of the operation of these two factors in Europe has been condensed into the last century, or even half-century, in the United States, especially in the centre and west of the country. The same phenomenon is marked in many of the British overseas possessions, and is likely to become increasingly evident in some in the future.

It is impossible to exaggerate the value to a working mycologist of a tour in the United States at the present time. In the last ten years the study of the diseases of plants in that country has progressed until it now easily leads the rest of the world. Since the War, American phytopathologists have redoubled their energies, and the next ten years will, I believe, see an accelerated progress. The most noticeable feature at the moment is the concentration of numbers of workers on certain fundamental problems, such as the etiology of the mosaic group of diseases, biological specialization in parasitic fungi, and the factors determining resistance or susceptibility to disease. Innumerable new points of view and critical attacks on many existing concep-

tions regarding parasitism and plant diseases generally are encountered, and a new science of phytopathology is being built up on the basis of the intimate relationship between host-plant, parasite, and external conditions. It is essential for the progress of mycological science in this country that we should keep in close touch with current developments and trends of thought in the United States. Mere knowledge of the literature does not sufficiently satisfy this need, as the value of published work is often difficult to appraise at a distance and trends of thought are usually in advance of publication. The practice of sending British mycologists to the United States in response to the annual invitations that have been issued during the past few years by the American Phytopathological Society is a sound one, and its cost will be more than repaid in the increased efficiency of their work.

To the Bureau of Mycology there is a special advantage over the general one referred to above, in that both as a centre for distributing information and an organization for working out the parasitic fungus flora of the outlying parts of the Empire we will require all the assistance we can get from other workers. As a result of my visit, I feel that we can rely confidently on receiving a great deal of help from workers in the United States.

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SOME CHARACTERISTICS OF THE VIRUS DISEASES OF PLANTS.

Being a Paper read in the Section of Pathology (Human and Comparative) at the Annual Meeting of the British Medical Association, Glasgow, July, 1922.

BY

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THE diseases of plants caused by an ultramicroscopic agent or virus appear to fall into four main classes. The largest is the group to which the mosaic disease of tobacco, first shown to be caused by a filtrable virus in 1892, belongs. The diseases in this group can be transmitted to healthy plants by grafting, in most cases (perhaps always) by sucking insects, usually by inoculating with the expressed juice or crushed tissues, and occasionally through the seed. There are no definite necrotic lesions, except in rare cases where necrosis of the parenchyma cells has been observed. They are not usually fatal, though often very harmful.

A second, small, group consists of diseases of which peach yellows is the type. Transmission has only been effected by grafting, but natural infection takes place in some unknown way, perhaps by insects. Transmission by inoculation with the juice or crushed tissues or by the seed has not been accomplished. No specific necrotic lesions are caused, but the cases are fatal.

A third group comprises the forms of variegation which can be transmitted to normal green plants by grafting. The affected plants are somewhat less resistant to unfavourable conditions than those that are normal, but the disease (if disease it can be called) is of a very mild type. Natural infection is not known.

Finally, there is a small group resembling the mosaic diseases in many respects, but characterized by definite necrotic lesions in that part of the vascular strands through which the elaborated food is conveyed to the growing or storage parts—the phloem. This group can be transmitted by grafting and also in some cases by insects, but not, so far as is known, by juice inoculations or through seed.

Transmission.

In all the cases investigated transmission can be effected by causing organic union between the tissues of a diseased and healthy plant, as by grafting or budding. In no case is mere contact sufficient. The next most general method is transmission by insects, this being common in the mosaic and phloem-necrosis groups, and possible, though not proven, in the peach yellows type. Transmission by inoculating with the juice of infected plants is only certainly known to occur in the mosaic group (much the largest). Transmission through the seed is rare, but has been fully established in certain mosaic diseases.

In transmission by grafting not every variety of plant that can be successfully grafted can be infected, though some of these diseases have a wide range of hosts. Sometimes, also, the virus is transmitted, but the new host shows no symptoms other than the ability to transmit the disease with full virulence by grafting on a susceptible host. Thus the phenomena of immunity and of "carriers" are known.

In transmission by insects usually there is no evidence that the insect is a necessary secondary host. Infection will occur if the insect is placed on a healthy plant immediately after feeding. In the "curly-top" disease of beet, however, infection cannot be conveyed for some time after feeding (four hours at high temperatures, up to forty-eight hours at lower temperatures). There is some evidence (as in several other allied diseases) that the infective agent is not always taken up with the plant juices by the insect, possibly because it is not always in a stage accessible to the latter. No other method of transmission than by the insect (*Eutettix tenella*) and by grafting is known. Evidently the causal agent must be an organism possessing a definite life history in host-plant and insect.

Spinach blight can be transmitted by several different insects and by juice inoculations. The incubation period in the plant varies according to the age of the plant and of the insect transmitter. The infective agent is hereditary in the insect up to at least four generations. The juice of infective aphids is infectious.

Transmission by inoculation with juice or crushed tissue has been most fully studied in the tobacco mosaic. The virus is present in all parts of the plant except the embryo of the seed. Infection can be produced by inoculating any part of the plant after abrading the cuticle. The leaf hairs of infected plants contain the virus, and it can be inoculated successfully by merely cutting first infected hairs and then those of a healthy plant with fine scissors. *Nicotiana viscosa* is immune to the mosaic of common tobacco (*N. tabacum*) but has a mosaic of its own, and both diseases will infect *Datura stramonium*. Carrier species are known—for example, *Physalis* can transmit the disease without showing any symptoms. Some species of aphids transmit readily, others fail though they feed and multiply on the plant.

In many mosaics the crushed plant tissue is more infective than the juice, but insects are often better than either. In some the virus is limited in distribution, and does not reach

the root, for instance. In a few cases biting insects (for instance, *Diabrotica* beetles) can carry infection.

Passage of the Virus within the Plant.

There is a good deal of evidence that the virus is conveyed chiefly through the phloem. This is practically the only tissue that shows necrosis in certain of these diseases. The most usual transmitting insects are aphids, and it is now known that many of these feed chiefly or exclusively by sucking the juices from the phloem. Translocation of starch (effected through the phloem) is impeded in several virus diseases. Removing the bark (including the phloem) around the shoots prevents the passage of the virus in the infectious chloroses. The failure to reach the embryo, which is such a marked character of many of these diseases, may be explained by the fact that the vascular strands terminate in the integuments and do not reach the embryo, which is nourished by osmosis from the tissues outside the embryo sac. In the cucurbits, in which mosaic is transmitted by the seed, there are special nutritive adaptations for feeding the embryo which may serve to explain its infection.

The passage of the virus is slow in the tobacco mosaic, where it may take over three days to traverse a leaf and its stalk. In cucumber mosaic a case is recorded where it took nine hours for the virus to traverse thirty inches of stem in an upward direction.

In the infectious chloroses there is evidence that though the virus may be distributed throughout the plant it only multiplies in the chlorotic areas. If these are removed the plant will ultimately give normal green leaves. But the virus persists in a latent condition in the dormant buds of such plants, and if these are forced into growth they give chlorotic shoots which reinfect the new growth of the whole plant.

Properties of the Virus.

The virus of tobacco mosaic filters through most porous filters. In one case filtration through the Chamberland filter gave 91 per cent. infection, through the Berkefeld normal 63 per cent., the Berkefeld fine 47 per cent., and the Kitasato 40 per cent. It is retained by the Livingstone atmometer porous cup and by seven-eighths of an inch of powdered talc. The whole of the virus in infective juice is carried down with the precipitate from 45 to 50 per cent. alcohol or with an aluminium hydroxide precipitate, without losing infectivity. Antiseptics give irregular results, mercuric chloride being sometimes less effective than copper sulphate for instance. Heating to 70° C. destroys the infectivity of some viruses, others may survive 90° C. Dried infective leaves may retain 100 per cent. virulence after three years, and bottled juice up to fifteen months, even when it putrefies. Evaporated juice is less resistant. Dilution to 1 in 1,000 does not reduce the infectivity, and 1 in 10,000 does not destroy it. Cucurbit mosaic virus is much less resistant than that of tobacco mosaic, and may not survive keeping for even a couple of days.

The optimum temperature for the activity of the tobacco mosaic virus, as judged by the length of the incubation period, is 28° to 30° C. and the maximum near 36°. No symptoms appear when cooled sufficiently to check growth of the plant or heated above 36°, but they reappear when normal temperatures are again used. In certain areas in Canada infected potato plants show no symptoms and give almost normal yields, so that climate may make a plant become a "carrier" of its own virus.

Only quite recently have bodies that may possibly be organisms been definitely observed in the cells of infected tissues. In the maize mosaic and that of *Hippeastrum*, Kunkel of Hawaii has seen bodies which he compares with the Negri bodies of rabies or with *Cytoryctes variolae*, but which from his figures also resemble Rickettsia in certain stages. In tobacco mosaic, Palm of Java has just reported the presence of bodies agreeing in every respect with Lipschütz's "Strongyloplasma" or the "Elementarkörperchen" of v. Prowazek, and in another form with Guarnieri's bodies (Cytoryctes). Neither author refers to Rickettsia, but there would seem to be evident resemblances.

Though the first demonstration that disease can be caused by a filtrable virus was made in a plant disease—tobacco mosaic—human pathologists have advanced the study of the possible causative agents beyond the stage reached by plant pathologists. The latter may, however, claim to have been among the earliest to have convicted insects of playing a primary part in natural infection; they have proved that these diseases can be hereditary in host-plant and in insect; and on the experimental side, owing to the naturally greater ease of experimenting with plants, have made them amongst the best known diseases. They have shown that the plant cuticle presents an impassable barrier to infection; that a saprophytic life of the organisms is highly improbable; and that they are often so closely associated in some way with the living tissues that infection is only possible when organic union between diseased and healthy tissues is effected. Finally, they are demonstrating the remarkable effect of the environment, especially temperature, on the course of some of these diseases.

is being realised that both the amphoteric and colloidal properties must be taken into account. Both of these last properties play a part in industrial applications; for example, the action of gelatin as a protective colloid is of great importance in the preparation of photographic emulsions; but in the operations of developing and fixing, its behaviour as an amphoteric substance must be considered, as may readily be realised when one remembers that the usual developers are alkaline and that acid fixing baths are often used; the swelling of the gelatin film will vary in the baths, and in the change from the developer to the fixing bath the gelatin must, at some time, pass through the isoelectric point (*compare* Sheppard, *Brit. Journ. Phot.*, 1922, in the press). Questions of the hardening and tanning of gelatin also arise, but these have not been touched upon in this article.

SOME CHARACTERISTICS OF THE VIRUS DISEASES OF PLANTS

By E. J. BUTLER, C.I.E., D.Sc., M.B.
Director, Imperial Bureau of Mycology

THE occurrence in plants of diseases caused by filterable viruses was first demonstrated by Iwanowski [1] in 1892 and fully established by Beijerinck [2] in 1898. Their studies showed that the juice of tobacco plants infected with the " mosaic " disease was capable of communicating the disease to healthy plants after passage through the Chamberland filter. Similar mosaic diseases are now known in a number of plants.

A little earlier than this, however, Erwin F. Smith [3] had investigated a very serious peach disease in the United States, and conclusively established the fact that, though no parasitic organism could be detected, a communicable virus was present in the diseased parts, and could be artificially transmitted to healthy trees provided organic union (as by budding diseased buds on healthy wood) was effected. Unlike tobacco mosaic, the virus of " peach yellows " has never been extracted and handled, nor is any other method of transmission established than that by organic union, though it is evident that other methods exist in nature since the disease is highly infectious and even has periods of epidemic outbreak. Whether the other two allied diseases of the peach subsequently described under the names " peach rosette " and " little peach " are regional effects of the same common cause or are distinct diseases is not certainly known.[4] The " peach yellows " type of disease has since been found in a few other cases, chief

[1] Iwanowski, D., *Bulletin de l'Academ. Imper. d. Science St. Petersbourg*, N.S., v, p. 67, 1892.

[2] Beijerinck, M. W., Over een Contagium vivum fluidum als oorzaak van de Vlekziekte der Tabaksbladen, *Verhand. Kon. Akad. van Wetensch. Amsterdam*, 2nd Sect., vi, 5, 1898.

[3] Smith, Erwin F., Peach Yellows: a Preliminary Report, *U.S. Dept. of Agric., Bot. Division, Bull.*, ix, 1888; Additional Evidence on the Communicability of Peach Yellows and Peach Rosette, *ibid., Division of Veg. Path. Bull.*, i, 1891.

[4] Blake, M. A., Cook, M. T., and Connors, C. H., Recent Studies on Peach Yellows and Little Peach, *New Jersey Agric. Exper. Stat. Bull.*, 356, 1921.

amongst which is the " spike " disease of the sandalwood tree in India.[1], [2], [3].

It has been known for more than a century that it is sometimes possible to communicate variegation of the foliage of plants to non-variegated plants in the same manner as that mentioned in the last paragraph, namely by grafting a variegated branch on a stock with normal green leaves. Subsequent work by Lindemuth,[4] Baur,[5] and others has established the existence of several examples of this communicable variegation, which Baur calls " infectious chlorosis." It is characterised by a deficient development of chlorophyll, but the plants are not sickly, though less resistant to unfavourable conditions than the normal individuals. The infectious types are not usually propagated by seed, though certain other quite similar variegations that are non-infectious are reproduced in the seedlings. Except for the innocuous nature of infectious chlorosis and the fact that spread to neighbouring plants does not occur in nature, so far as is known, there are obvious similarities between this condition and the group of diseases of which " peach yellows " is a type.

In none of the groups mentioned above has any definite lesion been detected in the tissues of the affected plants, except in one form of potato mosaic (" crinkle ") and in mosaic of maize, where some of the cells of the parenchyma may be affected. Arrested development of tissues or whole organs, localised hypertrophies, and malformations are not uncommon, but there is usually no definite necrosis of the cells in the earlier stages of the disease. In the later stages necrosis and death may take place—peach yellows, for instance, is invariably fatal —but this appears to be a generalised result of malnutrition, and not due to the local action of any specific toxin. There is, however, a group of diseases of the virus type characterised by a localised necrosis of a particular tissue, of which leaf roll of potato is the best known example.[6], [7], [8]. In this case there is a

[1] Barber, C. A., Report on Spike Disease in Sandalwood Trees in Coorg, *Indian Forester*, xxix, p. 21, 1903.

[2] Butler, E. J., Report on " Spike " Disease among Sandalwood Trees, *ibid.*, Appendix Series, 1903.

[3] Coleman, L. C., Spike Disease of Sandal, *Bull. Dept. of Agric. Mysore State, Mycol. Ser.*, No. 3, 1917.

[4] Lindemuth, H., Studien über die sogenannte Panaschüre, etc., *Landw. Jahrb.*, xxxvi, p. 807, 1907.

[5] Baur, E., in *Ber. d. Deutsch. Bot. Gesellsch.*, xxii, p. 453, 1904; xxiv, p. 416, 1906; xxv, p. 410, 1907; xxvi, p. 711, 1908.

[6] Quanjer, H. M., The Mosaic Disease of the Solanaceæ, its Relation to the Phloem-necrosis and its Effect on Potato Culture, *Phytopathology*, x, p. 35, 1920 [Bibl.].

[7] Schultz, E. S., and Folsom, D., Leaf Roll, Net-necrosis, and Spindling-sprout of the Irish Potato, *Journ. of Agric. Res.*, xxi, p. 47, 1921.

[8] Papers by Quanjer, Cotton, Murphy, and others in *Rep. Internat. Potato Conference of* 1921, Royal Hort. Soc., London, 1922.

definite pathological condition of that part of the vascular strands through which the elaborated food is conveyed to the growing or storage parts. The cell walls of the phloem swell and turn yellow and are altered in their chemical composition. Associated with this is an impairment of the function of the phloem characterised by failure to convey the normal food constituents from the leaves in which they are formed to the growing tissues in which they are required. The leaves remain choked with starch when those of normally functioning plants growing alongside are empty. Phloem-necrosis has actually been detected in only a few cases : in leaf roll of potato, sereh disease of sugar-cane, mulberry dwarf in Japan, mosaic disease of the sugar beet, the sieve tube disease of coffee in Surinam, and the brown bast disease of rubber. In certain other cases, however, particularly in peach yellows and the allied spike disease of sandal, there is such a marked interference with the translocation of starch from the leaves that there would seem to be some functional disturbance in the phloem, though hitherto no necrotic changes have been described.

Taking these four groups, and a few cases in which it is as yet uncertain whether the diseased condition is transmissible to healthy plants and if so in what manner, there are altogether some seventy or eighty genera of plants belonging to about thirty families which are affected by one or more of these diseases, and the number is rapidly being added to. As knowledge increases, it is probable that the rough classification here given will have to be modified : in particular the peach yellows type may be merged in the phloem-necrosis group.

Methods of Transmission

All of the cases that have been tested so far are transmitted to healthy plants by budding, grafting, or other organic union between diseased and healthy plants. Transmission by mere contact of uninjured surfaces has not yet been detected in any case.

The next most general method of transmission is by means of sucking insects, generally green-fly (aphids). This has not been established in the case of the infectious chlorosis group, and there appears to be no evidence that this type is transmitted in any other way than by organic union. On the other hand, in the peach yellows group, though no other method of transmission has been artificially accomplished than by organic union, there is the clearest possible evidence that natural spread occurs freely, and my own experiments in the case of spike disease of sandalwood lead me to believe that insects will ultimately be found to be responsible. In the other two groups,

comprising by far the greatest number of the diseases here referred to, insect transmission is fully established in a number of cases and is probably general.

Less common is transfer by direct inoculation with the juice of the infected plants. It is only certainly known to occur in the mosaic group. All attempts to secure infection in this way have failed in the peach yellows and phloem-necrosis groups, nor does there appear to be any evidence of it in the infectious chloroses.

Transmission through the seed is rare. It is only certainly known to occur in a few cases of mosaic diseases, *e.g.* in beans and clover, and occasionally in cucumbers, and in the mosaic-like disease of *Datura* mentioned in the next paragraph. It does not occur in most of the mosaic diseases tested, but is perhaps doubtful in one or more of the Solanaceæ affected with mosaic, especially one form of tomato mosaic.[1]

Amongst the most interesting cases of transmission by grafting is the recently described " quercina " disease of *Datura stramonium*.[2] It is fully infectious by grafting on healthy plants of this species, and spreads also in the field in some way unknown. Tested on a number of related species it was transmitted by grafting to a few in a mild form, producing little effect on the host. From the latter it could (in some cases at least) be transmitted back to healthy *Datura* plants. On immune species the virus failed to pass through as little as 3·6 cm. of the stem to reach a normal *Datura* graft in the stem near by. In the leaf roll of potato, also, allied species such as tomato can be infected by grafting, but the symptoms are less marked than on the potato or may even be entirely absent, though a graft is capable of transmitting infection back to the potato. Several such cases of " carriers " have been demonstrated in other virus diseases of the Solanaceæ. Even in the case of the infectious chlorosis group a similar phenomenon has been observed by Baur. When scions of an apparently immune species of *Abutilon* are grafted on *A. thompsoni* plants affected by chlorosis, they remain apparently healthy and show no symptoms. But they are capable of transmitting the disease to susceptible species grafted on them. On the whole, the examination of the experiments on transmission by grafting throws little light on the nature of these diseases, except that it gives room for speculation as to what the nature of a virus that requires organic union of the tissues for its passage can be. Possibly it is a parasite of the highly specialised type known in

[1] Westerdijk, Johanna, Die Mosaikkrankheiten der Tomaten, *Meded Phytopath. Labor. " Willie Commelin Scholten,"* i, 1910.

[2] Blakeslee, A. F., A Graft-infectious Disease of *Datura* resembling a Vegetative Mutation, *Journ. of Genetics*, xi, p. 17, 1921.

some plant rusts and mildews that needs the infected host cell to be alive and in normal health for its support and fails to maintain itself in weakened or dying cells. Wound infections, such as inoculation pricks, may fail in such cases by injuring the host cells, and only the infections of uninjured cells, with the establishment of symbiotic relations between living host and parasite, succeed.

The study of insect transmission is more helpful. Until it was discovered, there were many who held that no living organism need be invoked to account for the origin and infectious nature of these diseases, and there are still some supporters of the " enzymic " theory of mosaic disease who believe that the whole disturbance is due to nutritional disorders consequent on the introduction of some stimulus of a non-living nature that upsets the balance of the enzymic activities of the plant.[1] But the continued extension of some of these diseases to new areas, the rapid multiplication of the virus in the plant, its capacity to cause infection at very high dilutions sometimes exceeding 1 to 10,000, its power of unlimited transmission to new healthy individuals, its conveyance by insects and other points of similarity to certain virus diseases of animals, and above all, the discovery that the insect carrier may not become immediately infective after feeding on a diseased plant but requires a period of time before it is capable of transmitting the disease, have convinced most plant pathologists that, as Quanjer has recently put it in surveying these diseases, " One can hardly deny the existence of a whole world of ultra-microscopic organisms, causing them " (*Rept. Intern. Potato Conf. of* 1921, p. 138).

Undoubtedly the most important case in this connection is the " curly top " disease of beet, the first in which insect transmission was established.[2], [3]. The disease is only known to be transmitted by grafting and by the beet leaf-hopper, *Eutettix tenella*. After the latter has fed on diseased plants, it is not infectious for a period of from four to forty-eight hours. The shorter periods are obtained only at high temperatures. This is a clear case in which the infective agent requires to undergo some change or development in the insect transmitter before it can reinfect its plant host. If fed for only a short time on a

[1] Chapman, G. H., Mosaic Disease of Tobacco, *Massachusetts Agric. Exper. Stat. Bull.*, 175, 1917. Several of the recent German workers on the leaf roll and mosaic diseases of potato do not find it necessary to invoke the intervention of a living parasite in these diseases.

[2] Rand, F. V., and Pierce, W. D., A Co-ordination of our Knowledge of Insect Transmission in Plant and Animal Diseases, *Phytopathology*, x, p. 216, 1920.

[3] Severin, H. H. P., Minimum Incubation Periods of Causative Agent of Curly Leaf in Beet Leaf-hopper and Sugar Beet, *Phytopathology*, xi, p. 424, 1921.

diseased plant, the insect frequently fails to transmit the infection. It would appear that either the infective agent is not always in a condition to be taken up by the insect, or it is not always present in the tissues where the insect feeds, or possibly the insect itself is not always willing to feed in the proper way or is otherwise not always liable to take it up. On the other hand, a single leaf-hopper fed for five minutes on a diseased leaf has been recorded to be capable of causing the disease when transferred to a healthy plant. The disease can be transmitted to a number of other plants belonging to various families on which the leaf-hopper can feed, and some of these serve as a means of carrying the disease through the period of the year when there are no beets growing. The insect itself may remain infective for as long as 111 days after feeding.

In most cases that have been examined, however, the insect appears to be capable of transmitting the disease immediately after feeding, even though in several of these cases the juice of the diseased plant has not hitherto been found able to cause infection. The evidence in these last cases is not, however, very complete, and it is somewhat surprising that so important a question has been left unanswered in the case of the leaf roll and crinkle of potato, mosaic and leaf curl of raspberries, and the mosaic disease of the sugar beet. In these cases inoculations with the juice have failed, and if the insect transmitter does not become infective for some time after feeding, one would be led to suppose that there is a necessary part of the life cycle of the parasite in the insect, and so to understand why a direct transfer by juice is ineffective. Where direct inoculation by juice is successful, as in a number of the mosaic diseases (tobacco, potato, tomato, bean, clover, sweet pea, cucurbits, spinach, turnip, sugar-cane, etc.), it is evident that no essential part of the life cycle has to be passed in the insect host.

In spinach blight, a disease which appears to be of the mosaic type (though, unlike most other mosaic diseases, it is fatal), various interesting points in connection with insect transmission have been established.[1] It is carried by several insects (at least two species of aphids and the tarnished plant bug *Lygus pratensis*) and is also transmitted by direct inoculation with the juice of infected plants. The juice of the crushed aphids that have fed on diseased plants is infective. Infection can result if infective aphids are allowed to feed on healthy plants for only five minutes, and non-infected aphids can pick up the virus by feeding on blighted leaves for only ten minutes. Adult aphids are more infectious than those that are immature and the incubation period of the disease in the plant is shorter

[1] McClintock, J. A., and Smith, L. B., True Nature of Spinach-blight and Relation of Insects to its Transmission, *Journ. of Agric. Res.*, xiv, p. 1, 1918.

after infection by adults than by young. Several other conditions influence the length of the incubation period ; it is about twice as long, for instance, when the virus is taken from plants in an early stage of the disease than when taken from the later stages, though the percentage of successes on inoculation is about the same. After the aphid becomes infective it can carry the disease to several successive plants by feeding on each for a time. The progeny of the infected aphids kept from feeding on infected plants can transmit the disease up to at least the fourth generation. The infection is therefore " hereditary " in the aphid. The possibility that there is an incubation period in the insect before it can transmit the disease is not excluded. Non-infected insects that remained on blighted plants for different periods of time, from ten minutes to forty-eight hours, and were then transferred to healthy plants, caused symptoms of the disease to appear in the latter in from seventeen to twenty-four days, the incubation period being about the same in the case of those that fed fourteen, twenty-four, and forty-eight hours, but being longer in those that fed for two hours and ten minutes respectively.

The study of insect transmission has therefore shown that in curly top of the beet the insect is not a mere mechanical carrier of the infection, but is an alternate host in which the virus undergoes some change, and it has also shown that the infection in spinach blight is hereditary in the insect. These are important advances.

Transmission by the juices of infected plants has been most fully investigated in the mosaic disease of tobacco.[1] This is perhaps the most highly infectious virus disease of plants known, though it does not kill. The virus is present in all parts of the plant that have been found so far capable of separate examination—leaves, stem, roots, flowers, and seeds. In the latter it is present in the seed coats, but evidently not in the embryo, since it is not transmitted through the seed to the seedling. Direct testing of the embryo for the presence of the virus after removal of the enclosing tissues would probably not be possible without causing contamination. Even the hairs of the leaf contain it. Infection can be produced by inoculating any part of the plant, the slightest abrasion, even broken or cut leaf hairs, permitting entry. The leaf hairs of infected plants have been carefully cut with sterile scissors so as to avoid injuring the tissues below, and the scissors thus contaminated with the contents of the hairs have been used to cut the hairs of healthy plants and have caused these plants to become infected.

[1] Allard, H. A., The Mosaic Disease of Tobacco, *U.S. Dept. of Agric. Bull.*, 40, 1914, and subsequent papers in *Journ. of Agric. Res.*, v, p. 251, 1915 ; vi, p. 649, 1916 ; vii, p. 481, 1916 ; x, p. 615, 1917 ; and xiii, p. 619, 1918.

Rubbing the leaves with the fingers acts in the same way. With needle inoculations, single pricks often fail, the best results being got by multiple inoculations.

The virus does not seem to be equally active or equally concentrated in all parts of the plant ; thus inoculations from the roots give a lower percentage of success than those from the leaves. " Carriers " are known, *e.g. Physalis* may be inoculated and transmit the virus by subsequent inoculation from it to other plants, but itself may show no sign of the disease. So also the grafting of an infected plant on an immune variety of *Nicotiana* makes the juice of the latter infective, though no symptoms are produced. Direct inoculation of the virus into an immune species may set up a destructive tissue rot, though no other symptoms appear and though rotting is not a symptom of mosaic in susceptible plants. Possibly immunity here is related to those cases known in the plant rusts in which varieties whose cells are too readily attacked and killed are immune merely because the parasite is unable to feed and grow in the presence of dead cells, and soon dies out. Mixing the virus with the sap of immune species of *Nicotiana* does not reduce its virulence. The virus travels slowly in the plant. It has been found to take three days to traverse a leaf and its stalk. It can be transmitted to several other plants of the same family (Solanaceæ), including tomato, petunia, *Datura*, *Hyoscyamus*, and *Capsicum*, but the divergent results obtained by different investigators in Europe and America, together with other circumstances, indicate that there are two or more distinct mosaic diseases of tobacco, just as it is clearly proved that there are several of potato. *Nicotiana viscosum* is known to have a distinct mosaic disease not communicable to tobacco, though both it and the tobacco mosaic can be transmitted to *Datura stramonium*. Transmission by insects (aphids) is common, but not all insects that feed on tobacco can carry the virus. The large green aphis of lettuce (*Macrosiphon lactucae*) will feed and multiply readily on tobacco but does not transmit mosaic, nor does the white fly or red spider. Though the fresh juice is infectious, the best inoculum of all tried is the dried leaf tissue macerated in water.

The allied potato mosaic is not so readily transmitted as the last ; for instance, rubbing the leaves is not sufficient. Bean mosaic is also more refractory, as it is often difficult to get success by inoculating the juice into the tissues from a syringe or by rubbing the leaves ; the best results are got in this case by using the crushed tissue of the leaf. In the cucumber mosaic,[1]

[1] Doolittle, S. P., The Mosaic Disease of Cucurbits, *U.S. Dept. of Agric. Bull.*, 879, 1920, and The Relation of Wild Host Plants to the Over-wintering of Cucurbit Mosaic, *Phytopathology*, xi, p. 47, 1921.

too, the crushed tissue is somewhat more infectious than the extracted juice, but aphids are the most consistently successful agency for inoculation. In this case the virus is present in the stem, leaves, flowers, and fruit, but is apparently absent from the roots and usually from the seed. The leaf hairs contain the virus, and it can be successfully inoculated through broken hairs, just as in tobacco. Infection through the roots and blossoms has not succeeded. Seedling plants are less readily inoculated than those that are older but still growing rapidly. After growth has reached its maximum inoculation is again less often successful. The virus travels fairly rapidly through the plant. In one case in an inoculation at the tip of the stem, the juice of a leaf at the tip was infective twelve hours sooner than that of a leaf at the base of the plant; and in another case it took only about nine hours to traverse thirty inches of stem in an upward direction. Not only are practically all the species of Cucurbitaceæ tested susceptible to this disease, but it can be transmitted to members of several other families, including Solanaceæ, Compositæ, Campanulaceæ (*Lobelia*), and Asclepiadaceæ. Besides aphids it can be transmitted by beetles of the genus *Diabrotica*.

In the mosaic disease of sugar-cane, maize, sorghum, and various other grasses,[1] juice inoculations by injecting a considerable quantity of juice into the growing point have given some successes, but far less than when insects were used. The virus extends through the plant very slowly and healthy cuttings may be obtained from the lower part of the sugar-cane for an appreciable time after the top shows symptoms of the disease.[2] Both in maize and sugar-cane, new suckers may come up from diseased plants without showing any symptoms. There is also some evidence in the mosaic disease of potato that the virus reaches the tubers rather late in their development, and that it is at least sometimes possible to harvest immature tubers from diseased plants that may be used for "seed" without transmitting the disease.

The failure in most of the virus diseases of plants of transmission by the seed is chiefly of interest as indicating that, whatever the nature of the virus may be, it does not diffuse as readily from the supports of the ovule to the embryo as the organic nutrient materials do. In the quercina disease of *Datura* transmission by the pollen occurs as well as by the female gametes, and this appears to be the only case of the sort recorded.

[1] Brandes, E. W., The Mosaic Disease of Sugar-cane and other Grasses, *U.S. Dept. of Agric. Bull.*, 829, 1919, and Mosaic Disease of Corn, *Journ. of Agric. Res.*, p. 519, 1920.

[2] Lyon, H. L., Three Major Cane Diseases, etc., *Bull. Exper. Stat. Hawaiian Sugar Plant. Assoc.*, iii, p. 1, 1921.

Passage of the Virus within the Plant

So far the means by which the virus is conveyed from one plant to another have been outlined. The next point of interest is the consideration of the channels of carriage of the virus within the plant.

As already mentioned, practically the only gross changes of a necrotic nature, such as might result from the localised action of a parasite, that have been observed are in that part of the vascular strand, the phloem, through which elaborated nutrient material is carried. Even where no necrosis has been reported, the translocation of starch is impeded in several cases, such as peach yellows and spike disease of sandalwood, suggesting some disturbance in the phloem. Other observations tend to support the view that this is the main channel of extension within the plant. As a rule the most certain method of transmission of the disease from one plant to another is by the sucking insects, chiefly aphids, which can convey it. It has been shown in several cases that these insects feed by inserting their probosces into the phloem, usually of the finer veins ; other tissues are avoided, or tested and abandoned, until the phloem is reached. Again in the cucumber mosaic about twice as many inoculations succeed when the infective juice is pricked into the neighbourhood of the vascular bundles exposed when a leaf is torn off than when made into the stem at random. In the infectious chloroses girdling experiments have demonstrated that the virus is carried only through the bark, which in plants such as those worked with includes the phloem tissues, but not the other elements of the vascular ring. Furthermore, the symptoms after inoculation always appear first in just those parts to which the flow of the elaborated food is directed, namely in the young developing shoots and the like.

That the vascular system is in some way connected with the passage of the virus is made more probable by the frequent failure of the latter to reach the embryo. It is known that there is no vascular connection between the mother plant and the embryo, the bundles terminating either at the base of the ovule or spreading around its seed coats, but never penetrating the nucellus which surrounds the embryo-sac. Hence the latter is nourished entirely by osmosis. The embryo developing within the embryo-sac is still further removed from vascular connection with the mother plant. In the Cucurbitaceæ the nucellus is provided on the outside of its epidermis with a well-marked cuticle, and this epidermis persists in the ripe seed, though the rest of the nucellus is absorbed. The stalk end of the ovule (the chalaza) is suberified. Thus the whole of the

central part of the ovule is cut off from the seed coats and stalk by cutin or suberin. The embryo also, in the spherical stage, is provided with a complete investment of cuticle over its free parts. All the evidence available in regard to infection by the virus diseases indicates strongly that they are unable to pass through cutin and probably unable to pass through suberin (*e.g.* uninjured branches or a potato tuber). Hence one would expect that the virus of cucumber mosaic would fail to be carried over to the next generation in the seed. But this is one of the diseases which is sometimes transmitted by seed. The explanation of this anomaly is perhaps to be found in the work of Longo,[1] who has shown that since the cucurbit embryo is so completely isolated by impermeable layers, an adaptation of a curious nature has been developed in which the pollen tube opens a communication between the neck of the nucellus and the embryo-sac. Possibly the same channel could serve to transmit the virus to the embryo. Other nutritional adaptations for the absorption of organic food by the embryo, haustorial organs of the most varied morphological value, are known in a number of other families, and may perhaps have some significance in the inheritance of some of the virus diseases.

On the other hand, Allard [2] has shown that considerable interruption of the vascular tissue does not appreciably increase the time taken by the virus of tobacco mosaic (which is not inherited) to pass from the leaf to the stem, and he is therefore inclined to think it passes from cell to cell by diffusion, though the fine anastomosing lateral veins may aid. Doolittle [3] thinks the latter are the more important in cucumber mosaic.

In the infectious chloroses there is evidence that, though the virus may be distributed throughout the plant, it only multiplies in the chlorotic areas. If these are removed, as by defoliation or carefully cutting out all the yellow spots on the leaves as they appear, it is possible to get the plant to form only normal green leaves ultimately. But the virus can persist in a latent form in the dormant buds of such plants, and if these buds are forced to grow, they give chlorotic shoots which reinfect the new growth of the whole plant. The persistence of the virus in the latent condition in perennial root stocks of plants like *Phytolacca* and *Physalis*, in the swollen root of the beet, and in the potato tuber, is well known.

[1] Longo, B., Osservazioni e ricerche sulla nutrizione dell' embrione vegetale, *Ann. di Bot.*, ii, p. 373, 1905.

[2] Allard, H. A., Further Studies of the Mosaic Disease of Tobacco, *Journ. Agric. Res.*, x, p. 620, 1917.

[3] Doolittle, S. P., The Mosaic Disease of Cucurbits, *U.S. Dept. of Agric. Bull.*, 879, p. 39, 1920.

Properties of the Virus

Leaving the discussion of the means of transmission from plant to plant and the carriage of the virus within the plant, one may next consider some of the properties of the virus, which have been chiefly investigated by Allard and Chapman in the tobacco mosaic. In this case the virus can be filtered through the Chamberland, Berkefeld, and Kitasato filters without being retained, though it passes less readily through those with finer pores. Infectivity was 91 per cent. with the Chamberland, 63 per cent. with the Berkefeld normal, 47 per cent. with the Berkefeld fine, and 40 per cent. with the Kitasato, in one series of tests.[1] It is entirely checked by filtration through the Livingstone atmometer porous cup and through powdered talc $\frac{7}{8}$ inch thick. (The cucurbit mosaic virus filters through the Berkefeld, but not through the Chamberland filters tried.) The whole of the virus is carried down with the precipitate obtained by treating with 45 to 50 per cent. alcohol and with an aluminium hydroxide precipitate, without its infectivity being injured. It is quickly destroyed by alcohol of 75 to 80 per cent., formaldehyde of more than 1 to 800 destroys it in thirty-one days and 4 per cent. very quickly, copper sulphate readily destroys the infectivity of the virus under certain conditions, but mercuric chloride and carbolic acid have little effect. Heating to over 80° C. for five minutes sometimes destroys it, but it can sometimes withstand 90° C. for the same time. It is quickly killed near the boiling point. The dried leaves remain infective for very long periods, giving as much as 100 per cent. infection after three years. The bottled juice without preservative has remained highly infective for fifteen months, though it had undergone putrefaction, and the juice treated with toluol has been found infective after three years. The virus obtained from evaporated juice loses its infectivity more quickly. Dilution of the virus to 1 to 1,000 does not reduce its infectivity, and at 1 to 10,000 some infection can still be obtained, but higher dilutions generally fail. The virus of cucurbit mosaic behaves in exactly the same way in regard to dilution, but is much less resistant in other respects than that of tobacco mosaic. It is destroyed by formaldehyde, phenol, and copper sulphate in 0·5 per cent. solutions, and by mercuric chloride at a strength of 1 to 2,000. Heating above 70° C. renders it non-infectious and drying the infected tissue has also the same effect. The expressed juice is seldom infectious for more than

[1] Chapman, *loc. cit.*, p. 107.

forty-eight hours. In the bean mosaic [1] excised leaves retain their virulence for only about the same time, but the virus (which is transmitted by the seed) will survive any temperature that does not kill the seed. The virus of potato mosaic in the latent condition in the tuber requires apparently a longer time to kill by heat than the tuber itself does.[2] There are numerous scattered observations on various properties of the infective agent in several other mosaic diseases, but space does not permit of referring to them.

Influence of the Environment

The only other point of general interest in these diseases to which I wish to refer is the influence on them of external conditions. This is sometimes so marked as to have led to controversy as to whether the whole train of symptoms noticed in certain of these diseases may not be accounted for by unfavourable environmental conditions without invoking any parasitic agency. Cases in point are the spike disease of sandalwood, which has been proved to be graft-infectious, and the pecan rosette,[3] where no transmission from one plant to another has been proved.

In several cases the air temperature has been found to have a marked influence on the incubation period and symptoms of this class of disease. In the mosaic disease of tobacco the optimum for the activity of the virus, as judged by the length of the incubation period, is 28° to 30° C. and the maximum near 36°.[4] The development of the symptoms is slowed down by lower temperatures proportionately to the growth of the plant, and no symptoms appear at temperatures that quite check growth. Above 36° tobacco continues to grow, but the new growth in infected plants shows no symptoms of disease and even previously mottled leaves tend to become normal. The virus is not destroyed, however, as the symptoms reappear when lower temperatures are again employed. In cucumber mosaic [5] infection fails when the soil and air temperatures are below 20° C. Above this, the incubation period is shortened as 30° C. is approached. In raspberry mosaic,[6] leaves put out

[1] Reddick, D., and Stewart, V. B., Transmission of the Virus of Bean Mosaic in Seed, etc., *Phytopathology*, ix, p. 445, 1919.

[2] Blodgett, F. M., The Relation of Time and Temperature to the Killing of Potatoes and Potato Mosaic Virus, *Phytopathology*, xii, p. 40, 1922.

[3] Rand, F. V., Pecan Rosette, *U.S. Dept. of Agric. Bull.*, 1038, 1922.

[4] Johnson, J., The Relation of Air Temperature to Certain Plant Diseases, *Phytopathology*, xi, p. 447, 1921.

[5] Doolittle, S. P., Influence of Temperature on the Development of Mosaic Diseases, *Phytopathology*, xi, p. 46, 1921.

[6] Rankin, W. H., and Hockey, J. F., Mosaic and Leaf Curl of the Cultivated Red Raspberry, *Canada Dept. of Agric. Divn. of Bot. Circ.*, i, N.S., 1922.

during very hot weather show no symptoms. Total suppression of potato mosaic as a result of climate has been found to occur in parts of Canada,[1] so that affected plants give a practically normal yield, though they still retain the virus in an infective condition. Thus, under certain conditions the potato may act as a " carrier " of its own mosaic. In curly top of beet the interval after feeding on a diseased plant during which the insect is not infective is shorter at high temperatures than at low.

The influence of soil and fertilisers is marked chiefly in the direction that in good soils, and especially with heavy manuring, the symptoms may be masked and good yields obtained.[2] Peach yellows, spike disease of sandalwood, and pecan rosette are amongst the diseases in which the soil factor may be of importance.

Light may also have an influence on the development of symptoms. Baur showed that the contagium of the infectious chlorosis of *Abutilon* may be destroyed by growing the plants in darkness, and Chapman that tobacco plants exposed to blue light showed little symptom of mosaic, though the virus was still present.

General Observations

The fact that disease can be caused by a filterable virus was first demonstrated in a plant disease—tobacco mosaic. Human pathologists have, however, advanced the study of the possible causative agents beyond the stage reached by plant pathologists. Only two observers claim to have detected a possible organism in the cells of infected tissues in plants. In certain cells of necrotic areas in the pith of the maize stem and in the cells of the chlorotic parts of the leaves of maize and *Hippeastrum* Kunkel[3] has described bodies which he compares with the Negri bodies of rabies and with *Cytoryctes variolæ*. Some of his figures may perhaps remind one of certain appearances of *Rickettsia*. Palm[4] in Java has just reported the presence in the cells of tobacco plants affected with mosaic of bodies agreeing in every respect with Lipschütz's *Strongyloplasma* or the

[1] Murphy, P. A., Investigations of Potato Diseases, *Canada Dept. of Agric. Bull.*, 44, 1921.

[2] Perret, C., Sur les maladies des pommes de terres, *Ann. des Epiphyties*, vii, p. 304, 1921.

[3] Kunkel, L. O., A Possible Causative Agent for the Mosaic Disease of Corn, *Bull. Expt. Stat. Hawaiian Sugar Plant. Assoc.*, Bot. Ser. iii, p. 44, 1921, and Amœboid Bodies associated with Hippeastrum Mosaic, *Science*, N.S., lv, p. 73, 1922.

[4] Palm, B. T., De mozaiekziekte van de Tabak een Chlamydozoonose ? *Bull. Deliproefstat. te Medan-Sumatra*, xv, 10 pp., 1922 [English translation attached].

" elementarkörperchen " of v. Prowazek, and in another form with Guarnieri's bodies (*Cytoryctes*). Here, again, a resemblance to *Rickettsia* would seem to be indicated.

No systematic search for the agent in transmitting insects appears to have been made. That there is a gradation of size in the infective agent in the different filterable virus diseases of plants is evident from the filtration experiments, the cucurbit mosaic, for instance, being more easily removed by filtration than that of tobacco.

Various indications suggest that the parasite must often be closely associated with the living protoplasm of the host cells. The plant juice is not usually as highly infective as the crushed tissues, and transmission by grafting is one of the most successful methods and the only one known in several cases. There is not the slightest evidence that the infective agent can live saprophytically, and the failure to get infection through the soil in very many carefully conducted tests, though the underground parts of the plant are exposed to frequent wounding in many ways, is conclusively against a soil life. Failure to cultivate the active agent is common in the obligate plant parasites, none of the rusts (of which there are several thousands) having ever been grown apart from its host.

The localisation of active symptoms to developing parts of the plant is a remarkable feature of these diseases. Infection is followed by the appearance of symptoms (chlorosis and various other manifestations) only in the newly formed shoots and leaves. Those that have already reached their full size remain unchanged, though in many cases they are highly infective. The virus can at least live in the mature tissues, but it is apparently unable to do harm to any but developing tissues, except where it is able to cause necrosis, as in leaf roll disease of potatoes and allied diseases.

Though there is no evidence in many of these cases that the virus passes through any necessary part of its life cycle in the insect carrier, it is sometimes very noticeable that only certain species of insect can transmit it. In the tobacco mosaic some species of aphids will cause infection, but others fail, though they feed and multiply readily on tobacco; and in sugar-cane mosaic only *Aphis maydis* has been proved capable of transmitting infection, though many other sucking insects feed on cane. The case is possibly similar to that of Japanese river fever, where it would seem that the mite which ordinarily transmits the disease is not a mere mechanical carrier, but its intervention is not always necessary to secure infection, since all stages of development of the parasite can take place in man. Or we can imagine a condition similar to that obtaining in some heterœcious rusts, which can propagate indefinitely in the uredo

stage. In this case the insect would correspond to the æcidial host where the latter has ceased to be essential to the fungus, as with black rust of wheat in India. The similarity to typhus fever in many points is also worthy of note.

These diseases may be hereditary, both in the plant (transmission by seed in bean mosaic and the like) and in the insect (spinach blight).

The injurious effect of these diseases varies from almost nil to total destruction : sugar-cane mosaic in Louisiana produces such slight effects, even in cases of 100 per cent. infection, that no reduction in yield of sugar has been proved, while spike disease of sandalwood trees is invariably fatal and is exterminating this valuable tree throughout a large area. The disorder is clearly a nutritional one in the first instance and the profound alterations in the enzymic activity of diseased plants have been chiefly responsible for the so-called enzymic theory of mosaic disease. In a case like the potato, where Quanjer has recently distinguished no less than four distinct diseases of the infectious mosaic type, besides true leaf roll and a second leaf roll not yet known to be infectious, it is evident that an immense amount of work is still required before the effects on the metabolism of the host can be clearly understood.

The increasing attention being paid to this class of disease by plant pathologists is not entirely the result (though in great part) of improvement in diagnosis. In certain cases there is a real extension going on, as in the sugar-cane mosaic, which has appeared recently in several important cane-growing countries. It is not suggested that any of them are of recent origin, as the facts can be sufficiently explained by the great activity of recent years in the introduction and testing of new varieties of plants from all parts of the world. Sugar-cane mosaic has been present in the Dutch East Indies for many years, and there is little doubt that it has reached the New World with eastern sugar-cane varieties introduced in considerable quantity in recent years. As a class, the diseases mentioned in this paper rank at present amongst the most destructive diseases of plants known.

POPULAR SCIENCE

THE SCIENTIFIC PICTURES OF JOSEPH WRIGHT

By F. W. SHURLOCK, B.A., B.Sc., F.Inst.P.
Principal, Derby Technical College

Wright's three great scientific pictures, " The Orrery," " The Air-Pump," and " The Alchymist," accurately reflect the state of physical science in his day, and it is a very fortunate circumstance that they are now exhibited together in the Art Gallery of his native town.

Wright of Derby, as he is usually called, was born in 1734 and died in 1797, so that his working period coincides with the latter half of the eighteenth century. The physical science of the period, in contrast with that of our own times, was unspecialised and a matter of general interest among people of education. The number of educated people was, of course, smaller than in our own time, smaller too in proportion to the total population, the chief subjects of study being the classical languages and mathematics, but it was a happy feature that people with this training should nevertheless take a keen interest in the teachings of science. There seems to have been no tendency among the votaries of art to regard with antipathy the progress of science, and no tendency to decry the value of science in favour of the humanities. They were more leisurely days when the keenness of the struggle for existence and the inevitable tendency to minute specialisation, which necessarily restricts the scope of our intellectual interests, had not yet rendered it wholly unprofitable for any one man to take all knowledge for his province, but in an age when we have been not inaptly described as an unscientific people trying to live in a scientific age, one cannot help looking back with some regret to the more general interest in scientific knowledge of former times. In Joseph Wright we have a distinguished artist who was obviously an interested and careful student of contemporary science and whose knowledge of the scientific details portrayed in his pictures is surprisingly accurate.

Trans. Br. mycol. Soc., 10 (1-2) 119-121, 1924

THE CAUSE OF CITRUS SCAB.

(With Text-fig.)

By E. M. Doidge, Assistant Chief, Division of Botany, Pretoria, and E. J. Butler, Director, Imperial Bureau of Mycology, Kew.

It is now well known that the disease called scab or verrucosis of the lemon is not caused by *Cladosporium Citri* Massee as originally believed, but by another fungus which has been described as the causal organism by Fawcett*, Winston†, and others, but not named as its mycological position was not determined.

Scab is a common disease in rough lemons in the more humid parts of South Africa and the organism which produces it has been isolated a number of times during the past three years. Inoculations with a pure culture of this organism have invariably produced typical scabs on young lemon shoots kept in a humid atmosphere. Its cultural characters agree with those so fully described by previous workers, especially Winston; and the formation of true conidia, enabling the systematic position of the organism to be determined, has now been obtained.

The hyphae are of two kinds: (1) immersed or stromatic, conspicuously septate, often toruloid, up to 3 or 4 μ in diameter; and (2) superficial, creeping, obscurely septate, 1·5 to 2 μ thick, and uniform. The true conidia have been found only on the latter, terminally or on short, lateral, usually unbranched hyphae (Fig. 1). They are formed terminally on the narrowed end of the hypha, but the latter continues growth by a very fine branch arising a short distance below the apex (Fig. 3), so that the terminal spore and its stalk are pushed over to a lateral position. By the rapid repetition of this process small, elongated "heads" (Figs. 2 and 3) of conidia are formed, one (the youngest) being always terminal, and the others attached to lateral stalks. The conidia (Fig. 4) are hyaline, unicellular, oval, sometimes slightly narrowed at the basal end, and measure 2–6·5 × 1·3–2 μ, mostly 3–4 × 1·5–2 μ.

On the immersed hyphae (Fig. 5) lateral, bud-like branches are often formed, which readily become detached and continue to grow by a process of very irregular budding, mixed with the development of short hyphae. These bodies, which have been

* Fawcett, H. S. Citrus Scab. Florida Agric. Exper. Stat. Bull. 109. 1912.
† Winston, J. R. Citrus Scab: its cause and control. U.S. Dept. of Agric. Bull. 1118. 1923.

described by previous observers, are probably of an oidial chlamydospore nature and are not true conidia.

The fungus can be most satisfactorily placed in the form genus *Sporotrichum*, amongst the heterogeneous elements of which it most closely resembles the animal pathogens. This resemblance is apparent not only in the spore characters but

Sporotrichum Citri n. sp. 1. A sporiferous hypha with early stages of fertile branches. 2. A hypha with well-developed conidial heads. 3. Stages in the formation of the conidial heads. The upper figure shows renewed growth taking place a little below the conidium. 4. Conidia. 5. Immersed hyphae with bud-like detachable segments, and some of the latter germinating. All ×900.

also in the peculiar growth in culture, especially the raised, convoluted colonies, sometimes with an outgrowth of spiny synnemata, and the marked colour changes on different media. The pleomorphism of the human pathogen *S. Schenckii*

Beurmanni) is fully described by Davis*, and this paper, together with De Beurmann and Gougerot's monograph of the pathogenic species of the genus†, will serve to make the suggested relationship more clear. The specific name *Citri* is proposed (though the same or a closely allied species attacks the avocado pear), chiefly because the scab fungus has been so often referred to as *Cladosporium Citri* even after it was known that it was not a *Cladosporium* and was not the same as Massee's species. The following diagnosis has been drawn up from cultures.

Sporotrichum Citri Butl. n. sp. Mycelio superficiali ex hyphis repentibus, tenuibus, 1·5–2μ cr., obscure septatis, hyalinis, composito; conidiis pareis, initio acrogenis, solitariis, dein acropleurogenis, sporulas subcylindricas in ramis lateralibus mycelii formantibus distincte pedicellatis, hyalinis, ovalibus, basi subattenuatis, 2–6·5 × 1·3–2μ (saepius 3–4 × 1·5–2μ); mycelio immerso ex hyphis copiose septatis, 3–4μ cr., irregulariter toruloso-ramosis, hinc inde ramulos ellipsoideos, facile desciduos ac conidia simulantes gerentibus (?chlamydosporos), composito.

In foliis, fructibus, ramulisque vivis Citri.

NOTE ON THE ASSOCIATION OF TILLETIA TRITICI WITH "EPILEPTIFORM CONVULSIONS" IN THE DOG.

By J. Russell Greig, M.R.C.V.S., Professor of Medicine, Royal (Dick) Veterinary College, Edinburgh.

Although convulsions of an epileptiform character are commonly met with in the dog, their association with spores of fungi has not, so far as I know, been recorded.

Recently three cases, showing epileptiform symptoms, which appeared to be associated with the spores of *Tilletia Tritici*, were admitted to the hospital of the Royal (Dick) Veterinary College. The symptoms were identical in each case. The animal was suddenly attacked by a series of convulsions, which continued for some hours, and terminated in coma; in two cases death resulted, but in the other case recovery took place, though the animal was subsequently destroyed. As a routine measure the faeces in such cases are examined for the presence of Ascarid eggs, and in case A, although no eggs of parasites were found, the faeces contained very large numbers of spores, which proved to be those of *Tilletia Tritici*. An examination of the abdominal organs was made *post mortem*, but no definite abnormality was

* Davis, D. J. The identity of American and French Sporotrichosis. Univ. of Wisconsin Studies in Science, II, pp. 104–130. 1921.

† Les Sporotrichoses. Paris, 1912.

TROPICAL DERMATOLOGY.

Ota (Masao) & Langeron (Maurice). **Nouvelle classification des dermatophytes.**—*Ann. Parasit. Humaine et Comparée.* Paris. 1923. Oct. Vol. 1. No. 4. pp. 305–336. With 8 figs. [43 refs.]

Comparative studies of some fifty species of dermatophytes from authentic sources, belonging to the five genera usually recognised, namely, *Trichophyton*, *Microsporum*, *Achorion*, *Endodermophyton*, and *Epidermophyton*, have recently been made by one of the authors. It is stated that the existing classification of the dermatophytes into these five genera is not based on their mycological characters so much as on the nature of the lesions which they produce and is, therefore, botanically unsound. Numerous examples are cited in support of this view, which is one with which most mycologists will agree. Thus, morphologically, the *Trichophyton gypseum* group is stated to be much more closely allied to *Microsporum* than to most other species of *Trichophyton*; *Achorion annulosum* is nearer to *Trichophyton violaceum* than to *A. schönleini*; *A. gypseum* resembles the animal species of *Microsporum*; *Epidermophyton rubrum* is related to *T. gypseum* more nearly than to *E. cruris*; and so on. In reality, no morphological characters of weight serve to differentiate the five genera as at present recognised: all have approximately similar mycelial and chlamydospore characters; most have the peculiar type of lateral spore termed an "aleurie" by Vuillemin; and even the fusiform, pluricellular spores, which are perhaps the most characteristic feature of certain species, have been discovered in more or less perfect shape by the authors in *Achorion schönleini* and other forms in which they were not previously known to occur.

It is concluded that the dermatophytes form a homogeneous group, which is classed amongst the Hyphomycetes without prejudice to the possibility of their being conidial stages of Gymnoascaceae. Indeed, in view of the known production of skin lesions of the ringworm type by one of the latter family, *Eidamella spinosa*, and the formation of Gymnoascaceous perithecia in *Trichophyton currii*, this latter possibility is worthy of all consideration. It is supported by the authors' statement that *Gymnoascus reesii* has aleuria and fusiform spores of the same nature as those of *Trichophyton*, as well as by the occurrence of denticulate and spirally-coiled hyphae in several of the conidial dermatophytes.

The morphological characters of the mycelium and organs of dissemination are clearly described and illustrated. The mycelium is colourless or tinted reddish or violet. As is well known it has little taxonomic value, forms similar to those figured being found in many different groups of fungi: even the "nodular organs," which the authors are inclined to regard as rudimentary attempts at perithecium-formation, have, perhaps, no more significance than the mycelial "bulbils" found in other groups. The same is true of the so-called *Endoconidium*-type of conidia and the "arthrospores," which are merely oidia, chlamydospores, and resting or budding cells of the mycelium, similar to those known in many fungi. Possibly too much attention has been paid to these structures in the dermatophytes as a whole, and it can serve no useful purpose to dwell on them. The main organs of reproduction are the aleuria—unicellular, lateral conidia said to be usually liberated by the dissolution of the stalks connecting them to the

hyphae—and the fusiform spores, regarded as pluricellular chlamydospores. One may be permitted to doubt whether any of these structures have the morphological value of true conidia or whether they are not all merely of chlamydospore rank.

In view of the absence of clearly marked spore characters it is obviously far from easy to divide the mass of the dermatophytes into separate genera. Nevertheless the authors unite them into a separate subfamily of the Hyphomycetes which they term Closterosporeae, and then proceed to a new subdivision into six genera. It would surely have been more logically in accordance with their own observations regarding the scantiness of distinguishing morphological characters within the group to have united all into a single genus. The subfamily itself has little to commend it except as defining the essential unity of the dermatophytes. Of the older genera three are retained in an amended form, *Trichophyton*, *Endodermophyton*, and *Epidermophyton*, with the loss of certain of their species transferred to the other genera. Three new genera are created for the remaining forms. *Sabouraudites* contains the species co-generic with *Microsporum audouini*, Gruby (1843), and should by all the laws of nomenclature retain Gruby's generic name, notwithstanding that subsequent species have been wrongly placed in the genus. *Grubyella* is based on the forms co-generic with *Achorion schönleini*, Lebert (1845), and is an equally unnecessary creation. *Bodinia* is based on the type *B. violacea* (*Trichophyton violaceum*, Bodin, 1902) and is characterised by the general absence of any spore forms other than arthrospores (chlamydospores) in chains, though aleuria of a rudimentary type are occasionally formed.

With the views expressed in the earlier part of this paper one can have nothing but agreement. In the revised classification offered, however, it would seem that a golden opportunity has been lost of giving concrete form to these views by abolishing the artificial distinctions between the genera of earlier writers. Such a course might have done something to check the genus- and species-mongering that is the bane of medical mycology.

E. J. Butler.

Sabouraud (R.). **Sur la classification botanique des dermatophytes.**—*Ann. Parasit. Humaine et Comparée.* Paris. 1924. Apr. Vol. 2. No. 2. pp. 172–177.

Discussing the recently published classification of the dermatophytes by Ota and Langeron [see above] Sabouraud expresses his complete agreement with the view that all these organisms are closely related to one another and form a homogeneous botanical group. He also agrees that previous classifications had no more than a provisional value, based chiefly on clinical expediency. He is unable to accept, however, the method proposed by the authors in order to establish the morphological characters of the various dermatophytes, namely, their culture on a single type medium. He argues very truly that in the case of organisms such as these, admittedly particular in their nutrient requirements and often difficult to obtain in a freely-sporing form, the choice of a single medium is scarcely likely always to yield cultures in a full state of morphological differentiation. For a true botanical classification the organisms should be studied in the most fully differentiated condition, that is to say under optimum conditions for growth and sporing. In the absence of any such attempt he prefers to adhere to the present clinical classifications. From his long experience of the

dermatophytes he has come to certain conclusions as to the botany of these fungi. He considers that the human species of *Microsporum*, the animal species of the same genus, and the microid species of *Trichophyton*, form three sub-genera, but that certain other dermatophytes, *e.g.*, *Epidermophyton inguinale*, seem to belong to a distinct genus though still within the close family circle of the group.

E. J. Butler.

Lombardo (C.). **Di una particolare colorazione vitale di alcune dermatomiceti.** [Vital Staining of Dermatomycetes.]—*Giorn. Ital. Malattie veneree e della pelle.* 1923. Vol. 64. No. 2. pp. 720–721.

Application of chrysarobin to conditions of dermatomycosis causes a fairly definite staining of the tissues in 24 hours, becoming more intense later. Sections, after being washed in benzol and cleared in cedar oil, show the protoplasm of the fungi coloured a deep brown, owing, it is said, to oxidizing ferments in the fungi themselves. The author suggests the use of this process both for demonstration and identification of dermatophyta.

H. Harold Scott.

de Magalhaes (Octavio). **Apropos des teignes observées au Rio Grande do Sul (Brésil),** *Microsporum circuluscentrum.*—*C. R. Soc. Biol.* 1924. Jan. 18. Vol. 90. No. 1. pp. 53–55. With 2 figs.

Report of a case of T. tonsurans occurring in a child aged 18 months. Microscopical examination revealed a fungus in sparse amount with the appearance and arrangement of a microsporum. The cultures showed a concentric ringed formation of white, yellow and ash-coloured growth arranged excentrically about a raised snow-white downy central portion, the whole culture having an external white rayed border. In some cultures the yellow tint predominated to resemble the culture of *M. fulvum*. The microscopical appearances of the cultured fungus are described ; the absence of " fuseaux " is emphasized.

W. Jenkins Oliver.

Horta (Parreiras). **Uma nova tinha microsporica Brazileira.** *Microsporon Ramos*, **n. sp. (Nota previa).** [A New Species of Microsporon in Brazil].—*Brazil-Medico.* 1924. Feb. 2. Year 38. Vol. 1. No. 5. pp. 59–60.

Mention is made of three cases exhibiting a favus-like eruption of the hairy scalp, one of which was complicated by a pustular folliculitis. Growth of a microsporon was obtained on Sabouraud's saccharose medium. A yellow pigment was first produced in the culture and later a red pigment limited to the zone of growth and penetrating the medium. In some respects the organism resembled *M. ferrugineum*, but differed in the following points :—(1) The colour ; (2) *M. ferrugineum* shows no external spores, whereas they were abundant in the new species ; (3) Whereas *M. ferrugineum* resembles in culture *Achorion schönleini*, this resembles *M. equinum*. The author names the new species *M. Ramos* in honour of his colleague, Dr. João Ramos e Silva. A more detailed account with photographs is to appear later.

H. Harold Scott.

CARINI (A.). **Sur la dermatite verruqueuse.**—*Bull. Soc. Path. Exot.* 1924. Mar. 12. Vol. 17. No. 3. pp. 227–233. With 1 plate & 3 figs. [11 refs.]

Two cases of this dermatomycosis are reported from Brazil, of which the diagnosis of the first is based entirely on photographs of the affected area. The lesions of the second case were situated about the external malleolus and dorsum of the right foot, and consisted of papillomatous tumours raised 1–2 cm. above the skin surface covered with adherent thick crusts. Such papillomatous lesions, according to the patient, had developed from nodules of which type one was visible and was soft, fluctuating and contained pus. The affection dated from 16–17 years previously. All forms of treatment, including iodide of potassium, were without result. The pus on examination showed round and oval elements, 8–15μ in diameter, with a thick brown-coloured capsule. In addition there were distinguished occasional short mycelial filaments. Histologically the tumour resembled a granuloma with diffuse infiltration. The cultural characteristics are described. Probably the fungus had a saprophytic existence in the soil and became pathogenic through accidental inoculation of the skin. The fungus isolated from this case has not been identified. From previous cases reported two kinds of fungus had been denominated, *Phyalophora verrucosa* and *Acrotheca* respectively. The differential diagnosis is briefly discussed.

W. J. O.

LEÃO (A. E. Arêa). **Chromoblastomycose (Dermatite verrucosa mycotica. Observação de um novo caso).**—*Sciencia Medica.* Rio de Janeiro. 1923. Nov. 30. Vol. 1. No. 5. pp. 227–228. With 1 plate.

This is an account of a patient suffering from "Chromoblastomycosis" or verrucose dermatitis due to *Acrotheca pedrosoi*, differing from one previously recorded in which the condition was associated with dermal leishmaniasis.

A man, 63 years of age, resident for the past 15 years in S. Paulo, a labourer on the coffee plantations, where he worked barefoot: 12 years ago a single nodule developed on the left foot which increased from time to time till 3 years ago, when several fresh lesions appeared involving the whole foot, dorsal and plantar aspects, and the lower part of the leg. Each of these latter lesions was 1–3 cm. in diameter, with a distinct edge, raised, verrucose, crusted, reddish in colour, and exuding a blood-stained, seropurulent discharge. One larger lesion, 10 cm. in diameter, was present on the outer aspect of the leg at the junction of the upper and middle thirds. The patient was unable to walk on account of the pain. Sections showed a growth of the fungus Acrotheca and giant cell formation, while cultures made from material obtained by puncture yielded a growth of the organism.

An excellent photograph of the condition is given.

H. Harold Scott.

ROJAS (Hector M.). **Sobre la verdadera naturaleza del llamado "pie musgoso."** [The True Nature of so-called "Mossy Foot."]—*Cronica Méd.* 1923. Nov. Vol. 40. No. 725. pp. 361–381. With 8 figs. [4 refs.]

The author, after reviewing the recorded history of this condition, gives an account of two patients under his own care and of some experimental work in connexion with the disease. Mossy-foot, he

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Dr E. J. Butler, C.I.E. Bud-rot of Coconut and other Palms

A disease of palms, which was stated to have many points of similarity to that known as bud-rot in the West Indies, was described by the writer in 1906 and attributed by him to a species of *Pythium*, which was subsequently, in February 1907, named *P. palmivorum* n.sp. This was the first *named* cause of bud-rot which subsequent work has substantiated. The species is one of those forming a connecting link between the genera *Pythium* and *Phytophthora*, and in 1919, when knowledge of the latter genus had considerably advanced, the palm fungus was re-named by the writer *Phytophthora palmivora*.

In the West Indies the bud-rot of coconut palms seems to have been known for about a century, and was ascribed to various causes such as insects, bacteria, soil, and climate. Davalos believed that *Bacillus amylobacter*, which he isolated from the rotted tissues in 1886, was the cause in Cuba. Fawcett attributed it to "an organized ferment" in Jamaica in 1894. Earle also, in 1902, believed it to be a bacterial disease. In 1904 Dr E. F. Smith definitely stated, as a result of his investigations on the spot, that the disease in Cuba was a bacterial soft-rot of the terminal bud and its wrappings, the causal organism probably entering through wounds, though no experimental evidence of these statements was given. Petch stated, in 1906, that bud-rot in Ceylon was due to bacteria. Smith induced his assistant, J. R. Johnston, to undertake the detailed study of the disease, and the results of four years' work in Cuba and elsewhere were published by Johnston in 1912. Successful inoculations were claimed with several varieties of bacteria. Only a few of these inoculations were done on the spot, the rest being on seedling coconuts under glass in Washington. The greater number of the inoculations were made with *Bacillus coli* or an organism indistinguishable from it, while *B. coli* from animals was found capable of setting up a soft-rot similar to that caused by the strains from coconut, when inoculated on greenhouse seedlings. A preliminary account of these inoculations was published in *Phytopathology* in 1911. It may be mentioned, in passing, that the isolations were made by the poured plate method, which would be unlikely to reveal the presence of such a fungus as *Phytophthora*, and that the bulk of the inoculations were made with a gouge or some such instrument penetrating at least 30 cm. into the bud. But in 1911 Rorer agreed with Johnston's findings and stated that pure cultures of a

bacillus (which he isolated but did not identify) could cause bud-rot if poured into the crown without wounding.

In 1914 Shaw and Sundararaman published certain criticisms of Johnston's work and produced detailed evidence, founded on inoculations from pure cultures, that *Pythium palmivorum* (*Phytophthora palmivora*) caused coconut bud-rot in India. Further inoculation experiments, to meet objections raised by Sharples and Lambourne in Malaya, have been carried out by McRae and by Sundararaman in India, with the result that the earlier work was again confirmed. There is now not the slightest doubt that the epidemic outbreak of bud-rot in palms on the east coast of India was due to this fungus and only to this fungus, which is a true parasite capable of penetrating the uninjured tissues almost as readily as *P. infestans* does those of the potato.

In 1918 Ashby stated that there were two forms of bud-rot in Jamaica, one of which was due to the same *Phytophthora* as he had earlier found causing a leaf disease of coconuts. In 1920 this was identified as *Phytophthora palmivora*, the cause of the disease in India, and proof of its ability to penetrate the unwounded tissues was given. In the second type of bud-rot only bacteria were found, but some doubt is expressed as to their causal connection with the disease. In 1923, however, Nowell was still of opinion that after excluding the forms of bud-rot due to fungi there may still remain a large residue of bud-rot of bacterial origin in the West Indies.

In the Philippine Islands the study of coconut bud-rot was taken up by Reinking who, in 1918, believed it to be a bacterial disease. This view he soon abandoned and, in 1919, he stated that a *Phytophthora*, which he identified as *P. faberi*, was the cause, the bacteria which are always present in severe cases being secondary agents. In 1923 further studies were published by Reinking which seem to leave no room for doubt that this fungus is a true parasite and is capable of infecting uninjured coconut palms, whether the organism is isolated from coconut or from cacao (the original host of *P. faberi*).

This at once raises the question whether *P. faberi* is distinct from the earlier described *P. palmivora*. Reinking answers in the affirmative, but his argument is based on a misconception. No oospores were found in the Philippine fungus, whereas he states that they occur in that in India. It is true that in my 1907 paper I stated that oospores were formed, but these were subsequently found to be chlamydospores or resting conidia, of a type previously unknown in *Phytophthora* but subsequently found to be not uncommon. From a careful study of Reinking's figures

and description I am of opinion that his fungus is morphologically identical with my *P. palmivora*. Thus both in the west and in the east it has been established that this fungus is at least a chief cause of bud-rot.

As to Reinking's identification of this species with *P. faberi*, widely known as a parasite of cacao and rubber, a few words more are required. Ashby admits that *P. palmivora* and *P. faberi* are morphologically indistinguishable but, unlike Reinking, he finds biological differences. In his last paper on the subject, in 1922, he reports the discovery of oospores in mixed cultures of the cacao fungus with other morphologically similar strains from coconut and cotton and also with the distinct species *P. parasitica*. In view of the biological differences and the absence of oospores from all but the cacao strain he maintains *P. faberi* and *P. palmivora* as distinct species. But it is clear from Reinking's work that the biological differences are not preserved in the Philippines and it is equally clear that oospore-formation is rare and difficult to induce in the cacao fungus, so that I prefer to regard the two as at most strains of the one species, less highly differentiated indeed than many of the strains of the rusts and Erysiphaceae.

Whether there is any such thing as a primary bacterial bud-rot of palms seems to me to be doubtful. Most of the successful inoculations have been carried out through wounds in the tender tissues of the interior of the bud, and with such methods not one but several distinct bacteria have been found capable of setting up a soft rot. Sharples and Lambourne have indeed shown that various common moulds, such as *Mucor*, can cause such a rot in wound inoculations. The same is well known to be the case with ripe fruit and young seeds, always only after injury. I do not regard the fungus as a primary parasite in such cases, and it is at least in a very different category from the parasitism of such organisms as *Phytophthora infestans* or *P. palmivora*. *P. palmivora* remains the only vegetable parasite that has been proved to cause a destructive bud-rot of palms, capable of attacking perfectly healthy trees and inducing severe epidemics of disease.

[A short account of the campaign against bud-rot of palms on the east coast of India was then given.]

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METEOROLOGICAL CONDITIONS AND PLANT DISEASES.

In its early stages the study of plant diseases resolved itself into little more than a study of the parasites. Later, the study of the host plant received greater prominence, as the truth that disease is an indication of abnormal or disordered physiology became generally recognised. Still more recently has come the recognition of another truth, namely, that diseases caused by parasites are the manifestations of complex interactions of host and parasite under the influence of the conditions of a varying environment. Such conditions may be designated " environmental factors ".

No observant person who watches the incidence of plant diseases can avoid noticing the marked influence of one group of environmental factors, those included in what is rather vaguely termed the weather, on the prevalence of many of these diseases. To give only one instance, the unusual prevalence of the powdery mildews (Erysiphaceae) noticed in England in certain summers, such as that of 1921, is frequently ascribed to periods of dry, hot weather. At the same time, such a summer as that of 1921 was too dry for potato blight (*Phytophthora infestans*) to be severe in most parts of England. If these statements are true it is evident not only that disease is influenced by the weather, but also that different diseases are differently influenced by the same kind of weather.

Furthermore, if disease in any one country can vary from year to year under the influence of the annual fluctuations of the weather, we would expect to find that countries possessing different climates, that is different *average* weather conditions, would show still more marked variations in the prevalence of their plant diseases, owing to the greater amplitude of the variations between different climates than between those of the weather of any single climate.

It might be expected that comparison between the plant diseases of areas with different climates would be difficult because the host plants would not be the same. However true this may be for the wild plants, it is much less true in regard to the major cultivat-

ed crops. The latter have been subjected to long periods of artificial selection in such a way as greatly to extend their original range. Wheat and potatoes, for instance, are grown under very considerable differences in climatic conditions. Their parasites, on the other hand, have not been subjected to the same human influence, and they have had to follow the hosts as best they could. In the larger continental areas, such as the United States and India, the wheat, sorghum, maize, and potato areas are much greater than the areas in which certain of their parasites are found.

Weather and climate depend on the same set of factors — the ordinary meteorological factors of temperature, humidity, light, wind, etc. — and if it is desired to understand the relationship between meteorology and disease, an attempt must be made to evaluate the influence of each of these factors on the interaction between host and parasite. Probably this influence is capable, in most cases, of exact evaluation, but the data are as yet somewhat scanty. Temperature, humidity, and radiation are the chief factors about which data exist, and the following discussion will be confined to them.

The most difficult case is undoubtedly the study of the influence on disease of variations in weather conditions from one year to another within the same area. These variations are of lesser amplitude and duration than those between different climates, and most frequently are not large enough to prevent the disease from appearing but merely influence its amount.

In India rust is present every year in probably every wheat field. Whether an epidemic outbreak occurs or not appears to depend entirely on the weather conditions of the year. These conditions were analysed by MOREland (1) for 13 years in three selected districts in the mid-Gangetic Plain. He concluded that the deciding factor was the humidity of the air in January and February (the crop being sown in October and harvested about April), and that in the districts with the earliest harvest, the earlier part of this period was more important than the later. My own observations are in general agreement with these, except for the rather natural proviso that it is the humidity of the air within the crop that is of consequence. In a field at Pusa sown throughout with the same variety of wheat, rust (*Puccinia triticina* and *P. glumarum*) was severe in an area with a dense stand and almost absent towards the borders of the field where the growth was light. In the dense crop the air

humidity was nearly 20 % higher, four inches above the ground than in the light crop, as tested by an experiment lasting between 7 and 8 hours on a dull day. The temperature was about 3° C. lower in the dense crop. Soil moisture determinations showed that there was much more moisture under the dense crop, especially from the 3rd to the 6th foot, and this was really the cause of the good growth, as soil moisture is the chief limiting factor in wheat growing at Pusa (2).

Another disease, which at Pusa is dependent on humidity or rather on the deposition of dew is the die-back of chillies (*Capsicum*), due to *Vermicularia Capsici* (3). This disease appears at the flowering season about the beginning of October and may do very great damage. It ceases abruptly about 4 to 6 weeks later. This is because it is dependent on the heavy dews of this period, accompanied by a moderately high temperature. Artificial inoculations only succeed in almost saturated conditions. Plants growing under shade escape, as the deposit of the dew is slight. Later on, as the temperature falls and dew is less copious, the disease ceases. Observations during six years suggest that the relative air humidity requisite to cause an outbreak of this disease during the flowering period is over 85 %. In three of these years, when it was below this figure in the second half of September, there was no disease.

Potato blight (*Phytophthora infestans*) in the Gangetic Plain is influenced by a somewhat more complex group of factors. It is usually absent, and only two outbreaks have occurred in the last 25 years. Under certain circumstances a considerable portion of the seed used is brought down from the Himalayas where, at altitudes from about 4,000 to 7,000 ft., blight is endemic. If the Plains temperature is sufficiently high, infected seed thus brought down becomes sterilized, as it is well known that *Phytophthora infestans* is easily killed by heat. In 1912 a large import of diseased seed occurred rather late in the season, which was unusually cool at sowing time (October). In late December there was much fog and cloud with a low temperature, and an epidemic was in progress in January 1913 (4). Attempts to carry the fungus in culture over the following hot weather at Pusa failed and in the fields also there was no disease next year.

In localities where potato blight is endemic the problem is much more difficult and the meteorologic conditions that govern its intensity are by no means fully understood. It is one of the cases

in which a good deal is known, thanks to the work of Melhus (5) and others, of the conditions of temperature and humidity that influence the life-history of the parasite. The optimum temperature for the germination of the spores is between 10° and 13°C. A frost sufficient to kill potato leaves will also kill the mycelium of the fungus. Its upper temperature limits are below those required to injure the tuber, as it will not survive even 4 hours at 120°F., and cannot long be kept alive in cultures maintained throughout at over 90°F. But its optimum temperature for growth is much higher than the germinative optimum, being about 24°C. Its moisture relations are not so exactly known, but like all its allies it requires a high humidity for successful germination; it cannot stand drying for long, and zoospores are formed as a rule only in drops or films of water. The period of motility of the zoospores is longer at low temperatures; they can swim for 20 hours at 5°C., but only for 1 hour at 20°C. If the meteorological factors that influence the disease acted only by influencing the parasite, we would expect to find outbreaks always occurring when a cool, damp period alternated with a warm period at a time of the year, say July, in England when there is usually some blight in most of the potato fields. The cool period would assist germination and prolong the life of the zoospores, thus aiding dissemination, while the succeeding warm period would stimulate the growth of the fungus in the tissues. If another cool, damp period followed when the new infections were in the sporing stage, we would get the process repeated in an accentuated degree, and several such oscillations would enormously multiply the attack. But it is not easy to find records of such a regular sequence of events, partly because the meteorological data are not sufficiently detailed but perhaps also partly because the influence of the weather factors on the potato plant itself have not been studied. It is known, however, that in at least certain areas in the United States an unusually cool summer is necessary for severe attacks (6). It is also almost confined to the north-east, being seldom found below 40° of N. latitude. In Europe it is prevalent north of 50°N. In all cases it is worst where the humidity is high. In localities like the Channel Islands and the west of Ireland the humidity conditions are usually suitable, and temperature is likely to be the controlling factor in most years.

Many other diseases caused by Oomycetes appear to be influenced by rather similar conditions, as most of the Peronosporaceae have similar temperature relations. In order to demonstrate the

liberation of zoospores in the Phycomycetes it has been the custom in Pusa to use chilled water during the warmer parts of the year. The downy mildew of the grape, caused by *Plasmopara viticola*, appears to be an exception, as most observers consider that it is worst in relatively high temperatures.

It would appear that black rot of the grape vine, caused by *Guignardia Bidwellii*, is a similar case. It is stated that severe infection only occurs when there is a cold snap with rain, followed by a warmer period. Though the temperature relations of the fungus have not been worked out, so far as I know, this is very suggestive of their being similar to those just mentioned.

Another group of diseases that appears to be closely influenced by weather conditions is the powdery mildews (Erysiphaceae). It has already been mentioned that they are sometimes said to be worse in England in dry years. I have noticed the same thing in India with regard to the pea mildew, *Erysiphe Polygoni*. More exact observations are rare. Losch (7) examined the prevalence of apple mildew in an orchard situated on the slope of a ravine in Wurtemberg. The slope faced S. E. The upper part was severely attacked but, as one descended, the mildew decreased until at the bottom it was insignificant. The rays of the sun struck full on the upper slope. They reached lower down later in the day and, at the bottom, shade remained all the forenoon. The upper part was very hot and dry, the bottom cool and moist. No evidence was obtained as to whether the host or the parasite was most influenced, nor whether different varieties of apple would be equally affected.

Oak mildew behaves in the same manner. It has been noted (8) to be more severe in the upper levels of sloping woods, and also in exposed localities. Neger (9) has found that it produces its conidia much more vigorously on shoots exposed to the light than on those in the shade. Exposure and sunny weather favour the disease, but the relative importance of the factors of drought and radiation have not been fully estimated.

In Italy it has been noticed that either a drying out of the soil or a sudden rise in temperature makes wheat susceptible to *Erysiphe graminis*. Rivera (10) believes that this is due to a lessening of turgescence and is therefore a reaction of the host plant and not of the parasite. He says it is particularly marked in rich soils where the plants have a relatively restricted root development and would naturally suffer from shortage of water or excessive transpiration.

A hot sun and dry conditions are inimical to certain species of *Oidium* and epidemics are only likely to occur when there is a rapid alternation of moisture and dryness.

In India, tobacco mildew (*Erysiphe Cichoracearum*) is practically confined to shaded plants and the cereal mildew is also worse under shade (11). Most experimenters with cereal cultures kept indoors, must have noticed the greater prevalence of mildew under such conditions than in the open. More diffuse light and the absence of air currents appear to be the causes, and exposure to light and air the remedy. The same has been noticed in regard to the vine *Oidium*, amd those on strawberry and cucumber. These apparent discrepancies might disappear on more exact knowledge of the actions of the different factors. The latter have not been studied in detail as regards their effect on the host-parasite complex, but they appear to be sufficiently marked to permit of exact evaluation. It seems likely that their action on the host is of greater importance than on the parasite, contrary to what appears to be the case with the downy mildews.

All these are cases of parasites primarily of the aerial parts of the plants. If we turn to those of the roots we find rather more exact knowledge. The problem is perhaps easier, as the environment is relatively more stable.

The earliest, and still one of the most complete, studies of the influence of temperature on a soil-dwelling parasite was made by Balls in Egypt, in 1905-6, on the sore-shin disease (*Rhizoctonia Solani*) of cotton (12). The damage done by this disease is usually restricted to the first stages of development of the seedling. Once cork-formation begins damage ceases. Cotton is sown in Egypt at different times between the end of February and May. The earlier sowings may take about 12 days to appear above ground, while in the middle of April the seedlings may be up in 5 days. Growth of the cotton root increases with a rise of temperature, in an accelerated curve which ceases rather suddenly at about 37°C. The parasite — a fungus universally present in Egyptian soils — has a similar growth curve, ceasing very abruptly at about the same point, 37°C. It was determined with considerable probability, especially in the case of the fungus, that inhibition of growth at the higher temperature was due to auto-intoxication, of much the same type as the well-known staling phenomenon in bacterial cultures. The toxin is produced even at the lower temperatures, but is too slowly formed

to accumulate sufficiently to inhibit growth, unless the medium is scanty or the cultures are kept for longish periods, until the temperature approaches 37°. Then it is formed more rapidly than the fungus can stand, and growth ceases. When the fungus encounters the young tissues of the cotton seedling at a temperature of, say, 20°C., these are penetrated and the hyphae pass from cell to cell, destroying the tissues before the toxin has time to accumulate sufficiently to check their growth. But at temperatures approaching 33°C. auto-intoxication is more rapid and growth is delayed. At the same time, at this temperature (33°C.), the cotton plant has its vital activities near their optimum. Hence defensive cork-formation is vigorous and the parasite is checked after producing only a small scar. At 37°C. not even a scar is produced, growth of the fungus being entirely inhibited. Thus the late sown seedlings normally escape attack, but a cold spell of even a couple of days in May will cause the death of many of them. The seasonal prevalence of this disease in Egypt is therefore practically, a purely temperature reaction, affecting chiefly the parasite.

We can now leave the discussion of the influence of seasonal variations of weather on the incidence of disease, after noting that in few cases has there been a successful attempt to evaluate the influence of the different factors exactly. When we turn to those wider and more prolonged variations that constitute different climates, we find that greater success in this direction has been achieved.

In India a rice disease of great severity occurs in part of the great rice-growing tract at the head of the Bay of Bengal. It is not due to a fungus, but to a nematode worm (*Tylenchus angustus*) (13). Injury is confined to the parts of the plant above the water in which the rice is grown, and to reach these parts the worm must leave the water and climb up the plants. It is a pure ectoparasite, never entering the tissues. Experiments have proved that the worm is able to move on a dry surface, provided the relative air humidity is above some point between 90 and 95 %, probably near 93. Above this point it has the power to condense water, and it moves in the film surrounding it by a snake-like method of progression. Neither feeding nor reproduction are possible under totally immersed conditions, but only in damp air between 90 and 100 % relative humidity. Below 90 % it can neither move, nor feed, nor reproduce. Hence the disease is confined to areas of maximum air humidity during the summer and autumn months. It has been possible to correlate the

meteorological data from 6 reporting stations within the affected area, with the distribution of the disease and to forecast that the greater part of the rest of India, excluding parts of the Burma delta and some of the east coast districts, is not liable to become permanently infected.

The smuts of sorghum, particularly the grain smut (*Sphacelotheca Sorghi*), annually cause danage estimated at a million sterling in western India. They are also prevalent in the Punjab and submontane districts, but are unknown in the eastern half of the Gangetic Plain. KULKARNI (14) worked out the temperature relations of this disease. Sorghum has its optimum temperature for germination at about 37°C., at which temperature and suitable moisture the seedlings will be up in two days. At 20°C. they take about twice as long, and at 16°C. about three times. The spores of *Sphacelotheca Sorghi*, however, have their germinative optimum about 20° to 25°C., and germination falls off rapidly above 30°C., giving only 1 or 2 % at 37°C. Infection is only possible during the earliest stage of seedling development. At high temperatures not only is this stage shortened, but the smut spores germinate feebly. In incubator experiments no infection occurred at 40°C., whereas at 25° there was 50 to 60 %. In western India the main crop is sown at a time when the temperature is about 21° to 27°C., and naturally suffers severely. A second crop is often sown at a period when the temperature is over 30°C., and this suffers relatively little. At Pusa, in the eastern half of the Gangetic Plain, the temperature at sowing time ranges up to 38°C., and there is no natural sorghum smut, though by properly selecting the temperature at sowing time it can be artificially produced. In a comparative experiment seed grain was mixed with spores and divided into two lots. One sown at Jacobabad with an air temperature of 36° to 40°C. gave no smut. The other sown at Poona at 25°C. gave 65 % infection.

Wheat bunt (*Tilletia tritici* and *T. levis*) in India is a similar case. It is prevalent in Kashmir and along the Punjab foot-hills, occurs in the Punjab plains, but not severely, and is unknown further east or south. Bunt spores have a low optimum for germination (between 12° and 18°C.) and usually fail to germinate above 25°. In central and eastern India the temperature at sowing time is usually above 25°, while in northwestern India the temperature at sowing time is below. In the latter case it is low enough to permit the spores to germinate and infect the seedling, but further east and

south it is too high. HUNGERFORD (15), under Idaho conditions, found that the maximum degree of infection was obtained with a temperature of 9° to 12°C. and fairly high soil moisture.

Maize smut (*Ustilago Zeae*) has much the same distribution in India as bunt, but its temperature relations in that country have not been worked out. In America (16) it has been shown that the maximum for spore germination and the development of sporidia is between 36° and 38°C., so that it might be expected to be limited by high temperatures at sowing time in much the same way as sorghum smut, and its Indian distribution suggests that this is the case.

Thus there is a group of four of the major diseases of cultivated plants in India (potato blight, sorghum smut, wheat bunt, and maize smut) strictly limited in their normal distribution by the temperature relations of host and parasite, the influence on the parasite being the chief.

In the United States a great deal of information regarding the temperature and humidity relations of certain diseases has been accumulated during recent years. Only a few of the more striking cases, where a correlation with regional climatic conditions has been established, need be mentioned.

The seedling blight of maize and wheat caused by *Gibberella Saubinetii* is a severe disease in the United States, especially in the middle west. It was noticed that the southern part of the maize belt escaped this disease, as did also the northern part of the wheat belt. The temperature relations of the disease were recently worked out (17). Wheat and maize were grown in soil held constant at eight different temperatures from 8° to 36°C. and exposed to infection, with the result that the favourable soil temperatures for the infection of wheat were found to range from 12° to 28°C., the optimum being about 24°. Maize, on the other hand, was infected from 8° to 20°C., most severely from 12° to 16°. The reaction appears to be one of the host, not of the fungus (18). Like most soil *Fusaria* the latter has a relatively high optimum in culture (24° to 28°C.). At low temperatures in maize and at high temperatures in wheat, it was found that the cell-walls of the seedling long remained in an unthickened condition, easily penetrated by the fungus. At the higher temperatures in maize and at the lower temperatures in wheat, thickened cell, walls, which resist the fungus, are rapidly formed.

The temperature relations of a good many soil-dwelling parasites

have been worked out. In those attacking the potato, four species of *Fusarium* together with *Verticillium albo-atrum* have their growth optimum at about 25°, two species of *Fusarium* at 30°C. The latter two are the species that cause damage in the southern potato-growing region of the United States, *F. oxysporum* and *F. radicicola* (19). The cabbage yellows parasite, *F. conglutinans*, is one that requires a moderately high temperature to cause disease. At a soil temperature between 22° and 24°C., 97 out of 104 plants wilted, and between 10° and 16°, 1 out of 88 (20). *Fusarium Lini* infects flax chiefly between 20° and 30°C., and like the last disease its geographical distribution in America is limited accordingly (21). The tobacco wilt *Fusarium, F. oxysporum* var. *Nicotianal*, is the same, 28° to 31°C. being its optimum for infection (22), while little infection occurs at 15° or 34°. This limits its natural range to certain areas or to exceptionally hot seasons in other areas into which it may be introduced.

In these and many other cases the temperature relations of both host and parasite have been determined only for the underground system. Recently, however, it has been suggested that the air temperature to which the above-ground parts of the plant are exposed may be of importance. This has been established in the case of the *Fusarium* wilt of tomato (*F. Lycopersici*) (23). As in the case of the sore-shin of cotton the growth curves of the parasite and of the roots of the host are similar, the optimum being about 28°C. If the air temperature is held constant at 27° to 33°, fatal wilt develops at a soil temperature of 27° but not at 17° or 36°C. There is no external symptom of wilt if the air temperature is at 17°, no matter what the soil temperature may be, nor if the soil temperature is below 20° or above 34°, no matter what the air temperature may be, although both the host and the parasite can grow at these temperatures. Hence the disease is chiefly found, in America, in the Southern States and in England, in the hottest part of the year.

Onion smut (*Urocystis Cepulae*) is a similar case (24). According to the soil and air temperature used, onions can be grown with from 100 % to no smut as a result of inoculation. The reaction is believed to be chiefly one of the host plant. The seed will germinate over a wide range of soil temperature, from 10° to 31°C., but the best top growth is got with a soil temperature of about 20°C. Infection will occur at soil temperatures between 10° and 25°C., but little at 27°. At a soil and air temperature of 25° a great many plants will outgrow the attack, but at an air temperature even as

high as 30° to 33° infection can be got if the soil temperature is kept below 25°. In outdoor plantings infection was completely inhibited when the mean soil temperature reached 29°C. and increased steadily with lower temperature. This explains the geographical distribution of the disease in the United States, where it is absent from large areas in which onions are sown when the soil temperature is high.

Even mosaic diseases show definite temperature relations. In cucumber mosaic a high soil and air temperature favour the disease and the relations have been worked out in some detail by DOOLITTLE (25). JOHNSON (26) has given similar data in regard to the influence of air temperature on several mosaic diseases.

Humidity relations have not been worked out in so many cases in the United States.

In comparing oat rust (*Puccinia coronifera*) with mildew (*Erysiphe graminis*), FROMME (27) found that he could get no infection with the rust below 80 % relative humidity, and only 6 % at 93, taking infection at saturation to be 100. With the mildew, on the other hand, he got infection at from 75 to 80 % humidity.

LAURITZIN (28) found *P. graminis* on wheat still more restricted, as he got no infection at 92 % humidity and only succeeded at from 95 to 100 %. The same was required for *Colletotrichum Lindemuthianum* on beans, and nearly as high limits, 91 to 100 % for *Ascochyta Fagopyrum*.

Except in a few marked cases, such as potato blight (where temperature plays also a great part) and black rot of the vine, there has as yet been relatively little work done on humidity in relation to regional distribution. This is partly due to the greater complexity of the apparatus required for exact work. Thus, though we may believe that humidity is an important factor in the distribution of a disease in different climatic regions (as it certainly is in regard to its intensity in a given region) there is as yet little exact proof of this.

Still less have the finer details of the humidity relations of the host-parasite complex been worked out. There is just enough known to indicate that methodical research in this direction may give results of great interest. Thus PANTANELLI (29) has shown that infection of the vine leaf by *Plasmopara* is dependent on the condition of the stomata, being possible only when these are fairly widely open. Young leaves are immune because their stomata are

too narrow. So also in older leaves infection fails if the soil moisture is below 15 %, unless the air humidity is above 80 %. If the soil moisture is above 20 % the stomata open widely enough to permit infection at an air humidity above 40 %.

I have so far confined myself to the action of the individual factors on particular cases of disease. It would take too much space to examine climatic influences as a whole on the general characters of the parasitic flora of a region, such as the relative scarcity of the Erysiphaceae in the tropics. But one or two cases indicating the interest of general studies of this nature may be quoted.

In Tripoli, TROTTER (30) has made observations on the character of the fungus flora. It is a hot and dry area, ranging from a region of relatively small rainfall restricted to October to February near the coast, to a region of no, or only accidental, rain inland. Of 83 Basidiomycetes, in the wide sense, 62 are parasitic rusts and smuts, and of the whole fungus flora about 70 % are parasites. He suggests that the water requirements of the fungi have determined this altogether abnormal percentage of parasites, since the latter get their water from the host plants and are independent of the climatic shortage of water. Furthermore, species with a relatively short life-history predominate, especially the Deuteromycetes. Amongst the rusts an unusual number are short-cycle forms, and the same has been found for the Alpine rusts of Switzerland, the reason in both cases being the relatively short growth period that the climate permits.

Equally interesting data may be expected from ecological studies of the parasitic flora of different regions, a subject on which very little has been done. DUFRÉNOY (8) examined from this point of view the Barèges valley on the north of the Pyrenees. He studied the influence of altitude on the parasites of a certain number of plants found at all altitudes on the valley slopes, from the bottom to 2,000 m. He found three groups. In one, the parasite was equally prevalent at all altitudes (most Ascomycetes), in another it was more severe at the lower levels or confined to them (some rusts, potato parasites), and in the third it was found chiefly at the upper levels (oak mildew and some rusts). He concluded that radiation was the most important factor in influencing the vertical distribution of the parasites. Temperature was of importance in fixing the seasonal periodicity and the amount of damage caused to the host but had little effect on the presence or absence of the parasite.

The effects of shade were studied in some detail, and the chief interest in the work is the high importance given to the intensity and actinic power of the sun's radiation in influencing leaf parasites.

The practical interest and importance of the study of the relation between meteorological conditions and disease scarcely needs emphasis. In France (31) there has been for some time in operation a system of meteorological notifications from a central station at Montpellier, fed by some 60 recording substations, of the dates on which vine diseases are likely to appear and thus when spraying or sulphuring may be necessary. The notifications are based on a knowledge of the conditions influencing germination and infection, incubation in the tissues and subsequent sporulation, and are controlled by field observations at the Station. It is possible to predict sporulation seven days in advance, which gives ample time for protective measures. In Italy a similar service is operated from Turin (32).

In Germany the Biologische Reichsanstalt in Dahlem, Berlin, has established a laboratory for meteorology and phenology and has commenced the collection of data by means of a system of report cards issued throughout the country (33). A limited number of characteristic diseases are selected, and an attempt will be made to correlate their incidence and regional distribution with the isotherms and other meteorological data and with the annual fluctuations of the seasons. A search is also being made for indicator plants which may be found to have a reaction to seasonal conditions parallel with that of the parasites. HILTNER has already claimed that he can find a strict relation between the date at which snowdrops bloom and the extent of the plague of field mice which occurs from time to time in Bavaria.

The United States Weather Bureau is carrying on a large investigation to determine the effect of current meteorological factors on the growth of vegetation. As a part of this, systematic meteorological and phenological records are being kept at a number of centres with a view to determining the critical periods in the growth of crops and in the development of injurious insects and fungous diseases. Though the data are not as yet sufficient to yield much information regarding the incidence of diseases, some interesting correlations have been obtained, such as that potato blight makes its most rapid development when the daily average temperature is about 72°F. We have already seen that the optimum temperature of the parasite for growth in the host plant is about 24°C. or 75°F.

The object of this paper, however, is rather to stress the import-

ance of beginning at the other end —of first establishing the temperature and humidity relations of the parasite and host, singly and together, and only then, with the exact information thus gained, seeking the correlation with meteorological data.

The co-operation of the meteorological authorities will be required the moment it is sought to translate the results of such exact studies into practical application. Whether the meteorological data at present recorded are sufficient is doubtful. In India they are not, neither as regards temperature nor, still more, humidity. Even where the records are taken three times daily, a correction formula appears to be required. Several such formulae have been worked out for the mean temperature, but their application differ in different localities. For the purpose here indicated, not only the means but the duration of the daily extremes may be of the utmost importance, a low minimum, if sufficiently prolonged, being perhaps sufficient to start a parasitic attack, though it might not be revealed in the daily mean if counterbalanced by a hot day.

In this brief review many points of great interest, as, for instance, the fascinating problem of the overwintering of the cereal rusts, have been omitted. No attempt has been made to do more than give examples of various aspects of the influence of environmental factors on plant disease. Field observations on the seasonal and regional occurrence of diseases have, no doubt, a certain value, but the trend of recent work has been to show that definite conclusions cannot safely be drawn from such observations unless they are controlled and elucidated by the more exact methods of laboratory research.

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DER EINFLUSS DER METEREOLOGISCHEN BEDINGUNGEN AUF DIE PFLANZENKRANKHEITEN.

In den Anfangsstadien erschöpfte sich das Studium der Pflanzenkrankheiten noch fast rein in der Untersuchung der Parasiten. Später erlangte die Erforschung der Wirtspflanzen mehr Gewicht, als sich die Erkenntnis Weg bahnte, dass eine Krankheit die Folgeerscheinung physiologischer Störungen und Unregelmässigkeiten ist. Erst in jüngster Zeit ist man zu der weiteren Erkenntnis gelangt, dass parasitäre Krankheitserscheinungen als der Ausdruck einer Wechselwirkung zwischen Wirtspflanze und Parasit unter dem Einfluss verschiedener Umgebungsbedingungen anzusehen sind. Solche Bedingungen können als " Umgebungsfaktoren " bezeichnet werden.

Kein Mensch, der die Anzeichen von Pflanzenkrankheiten beobachtet, kann diese verhindern, wenn sie durch eine Gruppe von Umgebungsfaktoren bedingt sind, die mehr unbestimmt unter dem Namen Wetter zusammengefasst werden. So ist, um nur ein Beispiel anzuführen, das ungewöhnlich starke Auftreten des Getreideschimmels (*Erysiphaceae*) in England in manchen Sommern, wie auch 1921, vielfach trockenen, heissen Wetterperioden zugeschrieben worden. Gleichzeitig war ein Sommer wie der von 1921 in England für die Kartoffelfäule (*Phytophtera infestans*) zu trocken, als dass er viel hätte schaden können. Falls man sich auf die Berichte verlassen kann, geht klar aus ihnen hervor, nicht nur dass Pflanzenkrankheiten durch das Wetter beeinflusst, sondern auch, dass verschiedene Krankheiten durch die gleichen Wetterverhältnisse verschieden beeinflusst werden können.

Wenn weiterhin die Krankheiten in einem Lande von Jahr zu Jahr mit den jährlichen Wetterschwankungen sich ändern, so können wir annehmen, dass Länder mit verschiedenem Klima, d. h. mit verschiedenen Durchschnittswetterverhältnissen, noch viel ausgesprochenere Unterschiede in ihren vorwiegenden Pflanzenkrankheiten aufzuweisen haben, und zwar infolge der grösseren Schwankun-

gen im Klima der verschiedenen Länder gegenüber dem Klima ein und desselben Landes.

Man wird annehmen, dass ein Vergleich der Pflanzenkrankheiten von Gebieten mit verschiedenem Klima schwierig ist, da die Wirtspflanzen nicht die gleichen sein können. Dies trifft für die wildwachsenden Pflanzen zu, nicht aber für die Kulturpflanzen. Letztere sind durch lange Zeiträume bewusster Züchtung über ihren ursprünglichen Standort hinaus grossenteils zu allgemeiner Ausbreitung gelangt. Man denke nur an den Weizen und die Kartoffeln, die unter sehr verschiedenen klimatischen Verhältnissen angebaut werden. Andererseits sind ihre Parasiten nicht in solchem Masse dem menschlichen Einfluss unterworfen gewesen und den Wirtspflanzen nur so gut es ging, gefolgt. In weit ausgedehnten Kontinentalgebieten, wie in den Vereinigten Staaten und in Indien, erstreckt sich der Anbau von Weizen, Sorghumhirse, Mais und von Kartoffeln auf viel grössere Gebiete als bestimmte, sie befallende Parasiten angetroffen werden.

Wetter und Klima hängen von gleichartigen Faktoren ab, als da sind: Temperatur, Feuchtigkeit, Licht, Wind u. s. w. Wünscht man nun in das Verständnis der Wechselbeziehungen zwischen Meteorologie und Krankheitserscheinungen einzudringen, so muss man versuchen, den Einfluss eines jeden dieser Faktoren auf die gegenseitige Wirkung von Wirtspflanze und Parasit festzustellen. Wahrscheinlich werden bei genauerer Beobachtung die meisten Fälle auf ihren Einfluss zurückzuführen sein, doch fehlen vorerst noch genügende Angaben. Die meisten Angaben findet man über den Einfluss von Temperatur, Feuchtigkeit und Strahlung, und so sollen auch die folgenden Erörterungen auf sie beschränkt bleiben.

Die grösste Schwierigkeit bietet zweifellos das Studium des Einflusses der verschiedenen Wetterverhältnisse auf die Krankheiten auf dem gleichen Gebiet im Verlaufe eines Jahres. Die Schwankungen treten dabei nicht so stark in die Erscheinung, da sie kürzer dauern als bei dem Klima verschiedener Gebiete und da infolgedessen leichtere Beeinflussungen verhältnissmässig stärker in die Erscheinung treten, ohne dass sie genügend ausgeglichen werden.

In Indien tritt alljährlich fast auf jedem Weizenfeld Rost auf; es hängt lediglich von den jeweiligen Wetterverhältnissen ab, ob sich der Befall allgemein ausbreitet oder nicht. Die Bedingungen hiezu sind von MORELAND (1) im Laufe von 13 Jahren in drei ausgesuchten Distrikten der mittleren Gangesebene untersucht worden.

Er kam zu dem Ergebnis, dass der entscheidende Faktor im Feuchtigkeitsgehalt der Luft im Januar und Februar liegen müsse (wenn das Feld im Oktober bestellt und im April abgeerntet wurde) und dass die Ernte in den Distrikten, in denen sie am frühesten stattfand, bei dem früher geernteten Teil bedeutender war als bei dem später geernteten. Meine eigenen Beobachtungen stimmen im allgemeinen damit überein, nur dass ich annehme, dass es vor allem auf den Feuchtigkeitsgehalt der Luft zwischen den Pflanzen auf dem Felde ankommt, was auch näher liegt. In einem Feld bei Pusa, das mit der gleichnamigen Weizensorte bestellt worden war, trat Rost (*Puccinia triticina* und *P. glumarum*) auf einer dicht stehenden Fläche sehr ernst auf, während die lichteren Ränder des Feldes fast davon verschont geblieben waren. Aus einem Versuch, der an einem trüben Tag 7 bis 8 Stunden hindurch ausgeführt worden war, ergab sich, dass 4 Zoll unter dem Boden, wo die Pflanzen dicht standen, der Feuchtigkeitsgehalt der Luft fast 20 % mehr betrug als in derselben Tiefe unter den Pflanzen mit lichterem Stand. Die Temperatur war zwischen den Pflanzen mit dichtem Stand um etwa 3° C geringer. Untersuchungen der Bodenfeuchtigkeit wiesen einen viel höheren Feuchtigkeitsgehalt unterhalb der dicht bestandenen Stellen auf, besonders in einer Tiefe von 3-6 Fuss, und darin bestand offensichtlich der Grund für das gute Wachstum, da die Bodenfeuchtigkeit in Pusa der am meisten im Minimum befindliche Faktor ist. (2).

Eine andere Krankheit hängt noch in Pusa mit der Feuchtigkeit, oder besser gesagt mit der Betauung zusammen, nämlich das Abwelken der gemeinen Beissbeere (*Capsicum*), das durch *Vermicularia Capsici* (3) verursacht wird. Diese Krankheit tritt in der Blütezeit anfangs Oktober auf und kann sehr grossen Schaden anstiften. Ganz plötzlich verschwindet sie jedoch nach 4 bis 6 Wochen wieder. Der Grund dafür ist in dem starken Taubefall bei gleichzeitig ziemlich hoher Temperatur zu suchen. Die Erregung der Krankheit durch künstliche Impfung gelingt nur unter den Vorbedingungen, durch die sie auch sonst verursacht wird. Pflanzen, die im Schatten aufwachsen, werden nicht befallen, da sich auf ihnen der Tau nur schwach bildet. Später, sobald die Temperatur sinkt und der Tau nicht mehr so stark auftritt, verschwindet die Krankheit auch wieder. 6 jährige Beobachtungen haben ergeben, dass die relative Luftfeuchtigkeit, die für den Ausbruch der Krankheit während der Blüteperiode Vorbedingung ist, über 85 % betragen muss. In 3

von diesen Jahren betrug sie nicht soviel in der zweiten Hälfte des September, und die Krankheit trat auch nicht auf.

Die Kartoffelfäule (*Phytophtora infestans*) wird in der Gangesebene durch das Zusammenwirken mehrerer Faktoren beeinflusst. Für gewöhnlich tritt sie nicht auf; in den letzten 25 Jahren ist sie nur zweimal ausgebrochen. Bisweilen wird eine beträchtliche Menge Kartoffelpflanzgutes von den Himalayagegenden bezogen, auf denen die Fäule in Höhen von 4 000 bis 7 000 Fuss heimisch ist. Ist jedoch die Temperatur der Ebenen genügend hoch, so wird das Pflanzgut sterilisiert, da bekanntlich durch Hitze *Phytophtora infestans* leicht abgetötet werden kann. Im Jahre 1912 musste noch spät (im Oktober) befallenes Pflanzgut eingeführt werden, zu einer Jahreszeit, die für die Saatzeit ungewöhnlich kühl war. Gegen Ende Dezember war die Witterung bei niedriger Temperatur sehr neblig und trübe, sodass im Januar 1913 ein allgemeiner Befall eintreten konnte (4). Versuche zur künstlichen Erhaltung des Pilzes über die nun folgende heisse Zeit in Pusa waren nicht möglich, auch war im nächsten Jahr die Krankheit wieder verschwunden.

In Gegenden, in denen die Kartoffelfäule heimisch ist, liegt die Frage viel verwickelter und man hat sich noch keine volle Klarheit über die meteorologischen Bedingungen verschaffen können, die auf die Stärke ihres Auftretens Einfluss haben. Es liegt hier jedoch, dank der Arbeiten von MELHUS und anderen einer jener Fälle vor, bei denen man immerhin Etliches über den Einfluss der Temperatur- und Feuchtigkeitsbedingungen auf den Entwicklungsgang des Parasits kennt.

Das Temperaturoptimum liegt für die Sporenkeimung zwischen 10° und 13° C. Ein Frost, der das Abwelken der Blätter verursacht, genügt auch zur Tötung des Pilzmycels. Die Grenze des Temperaturmaximums liegt unterhalb der der Knollen, sodass diese dabei noch nicht beschädigt werden; der Pilz kann keine 4 Stunden 120° F überleben und auch nicht lange ausdauern, wenn die Kulturen ständig bei über 90° F erhalten werden. Das Temperaturoptimum für die Entwickelung ist jedoch viel höher als das für die Keimung; es liegt bei etwa 24°. Die Feuchtigkeitsbedingungen sind nicht so genau festgestellt, doch braucht der Pilz, wie alle anderen, viel Feuchtigkeit zu ihrem erfolgreichen Zustandekommen; Trockenheit kann er nicht vertragen; er bildet Zoosporen nur in Wassertropfen oder Häutchen. Die Bewegungsfähigkeit der Zoosporen ist bei niedriger

Temperatur länger: bei 5° C können sie 20 stunden lange schwimmen, bei 20° C hingegen nur eine Stunde. Angenommen, die krankheitbeeinflussenden meteorologischen Fakt'ren wirkten nur auf die Parasiten ein, so müsste man erwarten, dass ein Befall stets dann ausbrechen würde, wenn kühle, feuchte Perioden mit warmen zu einer Jahreszeit, z. B. Juli, wechseln, in der gewöhnlich die Fäule auf den meisten Feldern etwas zu finden ist. Die kühle Periode würde dabei die Keimung begünstigen und das Leben der Zoosporen verlängern, während die darauffolgende warme Zeit die Mycelbildung des Pilzes anregen würde. Wenn hierauf wiederum kühles, feuchtes Wetter folgte, so könnte man weiterhin annehmen, dass der Prozess von neuem, in verstärktem Masse einsetzen könne, wenn zuvor der Befall das Sporenstadium erreicht hätte, und so fort, so dass mit jedem weiteren derartigen Witterungswechsel ein immer steigender Angriff erfolgte. In Wirklichkeit werden jedoch Aufzeichnungen über eine so regelmässige Aufeinanderfolge der Erscheinungen wohl kaum zu finden sein, teilweise, weil die meteorologischen Daten zu wenig ins Einzelne gehen, teilweise vielleicht auch, weil der Einfluss der Wetterfaktoren auf die Kartoffelpflanze selbst noch nicht untersucht worden ist. Es ist jedoch bekannt, dass wenigstens in bestimmten Gegenden der Vereinigten Staaten nur ein ungewöhnlich kühler Sommer die Voraussetzung ernsten Befalls bildet. Dieser ist auch auf die nordöstlichen Gebiete beschränkt und tritt nur selten unterhalb 40° nördlicher Breite auf. In Europa ist er nördlich des 50. nördl. Breitegrades vorherrschend. In all den Fällen wird der Befall beträchtlich, in denen die Feuchtigkeit eine hohe ist. Auf den Inseln des Kanals und in Westirland sind die Feuchtigkeits-bedingungen dafür günstig gelegen, und es hängt dort in den meisten Jahren nur von der Temperatur ab, inwieweit die Krankheit auftritt oder nicht.

Viele andere durch die Oomyceten verursachten Krankheiten scheinen von ziemlich ähnlichen Bedingungen abhängig zu sein, da die meisten *Peronospora*-Arten ähnliche Anforderungen an die Temperatur stellen. Um die Abtötung der Zoosporen in den Phycomyceten zu bewirken, verwendet man in Pusa gewöhnlich gekühltes Wasser in den wärmeren Abschnitten des Jahres. Der Mehltau der Trauben, der durch *Plasmopara viticola* verursacht wird, scheint insoweit eine Ausnahme zu bilden, als er den meisten Beobachtungen gemäss in verhältnissmässig hoher Temperatur besiegt werden kann.

Es will scheinen, als ob der Fall bei der Schwarzfäule der Weintrauben, der durch *Guignardia Bidwellii* hervorgerufen wird, ähn-

lich liegt. Es steht fest, dass ernste Infektionen nur dann auftreten, wenn nach kurzer Zeit eine kalte, nasse Periode durch eine warme abgelöst wird. Obgleich die Temperaturverhältnisse dieses Pilzes noch nicht festgestellt worden sind, so liegen sie doch, soweit ich erkennen kann, den just erwähnten sehr nahe.

Eine weitere Gruppe von Krankheitserscheinungen, die anscheinend sehr unter dem Einfluss der Wetterverhältnisse stehen, werden durch den Mehltau (*Erysiphaceae*) hervorgerufen. Es ist bereits erwähnt worden, dass sie in trockenen Jahrgängen in England schlimmer als gewöhnlich auftreten. In Indien habe ich das gleiche beim Schimmel der Leguminosen (*Erysiphe Polygoni*) bemerken können. Genauere Beobachtungen finden sich spärlich. Losch (7) untersuchte den Befall des Apfelmehltaus am Abhang eines Hohlweges in Württemberg. Der Abhang lag nach Südosten. Der obere Teil war ernstlich befallen; tieferhin nahm der Mehltau jedoch ab und wurde schliesslich am Wegrand fast unmerklich. Die Sonnenstrahlen konnten den oberen Hang unbehindert treffen, tiefer unten kamen sie erst im Laufe des Morgens hin, und der Wegrand blieb den ganzen Vormittag im Schatten. Der obere Teil war recht heiss und trocken, der Wegrand kühl und feucht. Es war jedoch nicht klarzulegen, ob die Apfelbäume oder der Parasit am meisten beeinflusst worden waren, noch welche verschiedenen Sorten in gleichem Masse befallen werden.

Der Eichenmehltau verhält sich in gleicher Weise; er ist (8) in höher liegenden Gebieten abschüssiger Wälder und an ausgesetzten Stellen stärker bemerkt worden. Neger (9) hat beobachten können, dass er seine Conidien in weit stärkerem Mass auf Schösslingen bildet, die dem Licht ausgesetzt sind, als auf solchen, die im Schatten liegen. Ausgesetzte Lage und Licht begünstigen die Krankheit, doch ist die Bedeutung der Dürre- und Belichtungsfaktoren bisher noch nicht genügend festgestellt worden.

In Italien hat man beobachten können, dass sowohl Austrocknung durch die Sonne als auch plötzliche Temperaturerhöhungen den Weizen für *Erysiphe graminis* empfänglich machen. Rivera (10) nimmt an, dass diese Tatsache auf die Schwächung des Saftdrucks zurückzuführen s. i. Es sei darum die Pflanze selbst, nicht der Parasit, die erste Ursache für die Krankheit; vor allem sei dies auf reichen Böden augenfällig, auf denen die Pflanzen eine verhältnismässig geringe Wurzelentwicklung aufzuweisen und darum unter Wasserknappheit oder Wasserausdünstung viel stärker zu leiden hätten. Starke Sonnenbestrahlung und Dürre sind hinwiederum bestimmten *Oidium-*

Arten schädlich; diese nehmen nur dann überhand, wenn Feuchtigkeit und Dürre sich in raschem Wechsel ablösen.

In Indien ist der Tabakmehltau (*Erysiphe Cichorarum*) unter Schattenpflanzen verbreitet und auch der Getreideschimmel tritt im Schatten schlimmer auf (11). Viele Versuchsansteller haben feststellen müssen, dass Getreidekulturen in abgeschlossenen Räumen viel stärker von Schimmel befallen werden als im Freien. Mehr zerstreutes Licht und Mangel an frischer Luft scheinen ihre Ursache, Licht- und Luftzufuhr dagegen das Heilmittel dafür zu sein. Dasselbe ist beim *Oidium* des Weines, der Erdbeere und der Gurken beobachtet worden. Diese anscheinenden Widersprüche lassen sich jedoch bei genauerer Kenntnis des Einflusses der verschiedenen Faktoren lösen. Diese sind zwar hinsichtlich ihrer Wechselwirkungen zwischen der Wirtspflanze und dem Parasit noch nicht eingehend untersucht worden; doch genügen die vorhandenen Kenntnisse, um Schlüsse aus ihnen ziehen zu können. Es scheint, dass ihr Einfluss auf die Wirtspflanze grösser ist als auf den Parasit im Gegensatz zu dem auf den Mehltau.

All dies sind Fälle von vornehmlich oberirdischen Pflanzenparasiten. Wenden wir uns den an den Wurzeln zu, so finden wir hier viel genauere Kenntnisse. Die Frage liegt dabei insofern vielleicht einfacher, als die Umgebungsbedingungen hier im Durchschnitt gleichmässiger sind.

Eine der ersten und bisher vielleicht besten Arbeiten über den Einfluss der Temperatur auf einen Bodenparasiten wurde von Ballis in Aegypten von 1905-06 unternommen, u. zw. über die Rhizoctoniafäule (*Rhizoctonia Solani*) an Baumwollpflanzen. Der Schaden, der durch diese Fäule verursacht wird, beschränkt sich meist auf die ersten Entwickelungstufen der jungen Pflänzchen. Sobald die Verkorkung einsetzt, hört die Fäule auf. Die Baumwollsaat wird in Aegypten zu verschiedenen Zeiten zwischen Ende Februar und Mai vorgenommen. Frühe Saat geht erst nach etwa 12 Tagen auf, während bei der von Mitte April schon nach 5 Tagen die Keimpflänzchen hervorkommen. Das Wachstum der Baumwollwurzel steigt mit der Temperatur in ziemlich steiler Kurve bis zu etwa 37° C, bei der es plötzlich aufhört. Der Parasit, der auf den Böden Aegyptens allgemein verbreitet ist, weist eine ähnliche Entwickelungskurve auf, die ebenfalls bei 37° C plötzlich unterbrochen wird. Es wurde besonders bei diesem Pilz ziemlich genau festgestellt, dass diese Entwickelungstörung bei höherer Temperatur auf Selbstvergiftung

der gleichen Art zurückzuführen ist, wie sie bekanntlich bei den Bakterienkulturen in die Erscheinung treten kann. Das Gift wird auch in niedrigeren Temperaturen, doch langsamer ausgeschieden, sodass es sich nicht genügend ansammeln kann, um die Entwicklung zu verhindern, es sei denn die Nährlösung würde spärlich vorhanden sein oder die Zeit der Kulturen würde verlängert werden, bis diese die Temperatur nahe an 37° C erreicht hätten. Dann würde das Eigengift schneller erzeugt als es der Pilz vertragen kann, sodass er sich nicht mehr vermehren kann.

Wenn der Pilz bei etwa 20° C auf junges Gewebe der Baumwollkeimpflanze stösst, so durchdringt er es mit seinen Hyphen, die von Zelle zu Zelle vordringend die Gewebe zerstören, noch ehe das Gift ihrer Ausscheidungsstoffe sich genügend ansammeln kann, um ihr Wachstum anzuhalten. Nähert sich die Temperatur 33° C, so schreitet die Selbstvergiftung schon rascher vor sich und hemmt die Pilzentwickelung. Gleichzeitig erreicht dabei (33° C) die Baumwollpflanze beinahe ihr Optimum der Entwickelung. Von nun an geht die abwehrende Korkbildung rasch vor sich, sodass der Pilz sich nicht mehr rühren kann und nur mehr kleine Wundstellen bildet. Bei 37° C wird der Pilz vollständig unterdrückt. So entgehen spät gesäete Keimpflänzchen für gewöhnlich dem Befall; ein Witterungsrückschlag im Mai jedoch, selbst wenn er nur wenige Tage dauert, vernichtet viele von ihnen. Das an die Jahreszeit geknüpfte Auftreten dieser Krankheit in Aegypten ist also eine reine Temperaturwirkung, u. zw. hauptsächlich auf die Parasiten.

Wir wollen nun die Betrachtung über den Einfluss der durch die Jahreszeit bedingten Wetterschwankungen auf den Krankheitsbefall verlassen, doch sei noch darauf hingewiesen, dass man in einigen Fällen die ziemlich genaue Feststellung des Einflusses der verschiedenen Faktoren durchgeführt hat. Wenn wir uns nun den ausgeprägteren, grösseren durch die Klimaunterschiede bedingten Verschiedenheiten zuwenden, so werden wir bemerken, dass in dieser Richtung mit mehr Erfolg gearbeitet worden ist.

In Indien tritt in dem grossen Reisbaugebiet des Bengalischen Meerbusens eine sehr ernste Reiskrankheit auf. Sie wird nicht durch Pilze, sondern durch Nematodenwürmer (*Tylenchus angustus*) (13) hervorgerufen. Der Angriff erfolgt auf die über dem Wasser befindlichen Teile der Pflanze, zu welchem Zweck die Nematodenwürmer das Wasser verlassen und an den Pflanzen emporkriechen müssen. Sie sind nur äussere Parasiten, die niemals in die Gewebe

eindringen. Versuche haben gezeigt, dass der Wurm imstande ist, sich auch auf trockener Oberfläche fortzubewegen, vorausgesetzt, dass die relative Luftfeuchtigkeit über einer bestimmten Grenze zwischen 90 und 95 %, wahrscheinlich um 93 %, liegt. Oberhalb dieser besitzt er die Fähigkeit, das Wasser zu kondensieren und bewegt sich in einem daraus gebildeten Häutchen nach Schneckenart vorwärts. Bei völliger Unterwassersetzung der Reisfelder kann der Wurm sich weder ernähren noch vermehren, sondern nur in feuchtigkeitsgesättigter Luft mit 90 bis 100 % relativer Feuchtigkeit. Unter 90 % kann er sich wiederum weder fortbewegen, noch ernähren, noch fortpflanzen. Die Krankheit ist also auf Gebiete mit einem Maximalfeuchtigkeitsgehalt während der Sommer- oder Herbstmonate beschränkt. Es war möglich, meteorologische Daten aus 6 Berichtsstationen innerhalb des befallenen Gebietes mit Angaben über die Verbreitung der Krankheit untereinander zu vergleichen und zu schliessen, dass der grösste verbleibende Teil Indiens mit Ausnahme der Gebiete des Burma-Deltas und einiger an der Ostküste, einer ständigen Gefahr des Befalls nicht ausgesetzt sind.

Die Russbrand-Arten der Sorghumhirse, besonders der Kornruss (*Sphacelotheca Sorghi*), verursachen jährlich in den westlichen Teilen Indiens Schäden, die auf eine Million Pfund Sterl. geschätzt werden. Sie treten besonders in den Punjab- und in den Vorgebirgsgebieten auf, sind hingegen in den östlichen Teilen der Gangesebene unbekannt. KULKARI (14) hat die Temperaturverhältnisse dieser Krankheit untersucht. Das Temperaturoptimum für die Keimung liegt bei der Sorghumhirse um 37° C; bei dieser Temperatur und bei genügender Feuchtigkeit gehen die Keimlinge in zwei Tagen auf; bei 20° C brauchen sie etwa zweimal, bei 16° C etwa dreimal so lang. Die Sporen von *Sphaceletheca Sorghi* haben dagegen ihr Keimoptimum zwischen etwa 20° und 25° C; bei 30° fällt die Keimkurve plötzlich ab und zeigt bei 37° C nur mehr 1-2 % Keimfähigkeit. Die Ansteckung ist nur in der frühesten Entwickelungsperiode des Keimlings möglich; bei höherer Temperatur wird nicht nur diese abgekürzt, sondern auch die Keimung der Brandsporen erfolgt schwach.

Bei Ansteckungsversuchen erfolgte bei 40° C kein Befall, hingegen jedoch bei 25° C zwischen 50-60 %. In den Westgebieten Indiens wird die Hauptaussaat zu einer Zeit vorgenommen, in der die Temperatur etwa 21°-27° C beträgt, wodurch sie natürlich schwer gefährdet ist. Ein zweites Mal säet man, wenn die Temperatur über 30° steht; die dabei entstehenden Keimpflanzen werden verhält-

nismässig wenig angegriffen. In Pusa, im östlichen Teil der Gangesebene, liegt die Temperatur bei der Aussaat über 37° C, auch tritt hier tatsächlich Sorghumbrand nicht auf, es sei denn, dass man die für den Befall günstige Temperatur ausdrücklich abpasse. In einem vergleichenden Anbauversuch wurde das Saatgut mit den Sporen vermischt und in zwei Teile geteilt; der eine wurde in Jacobabad bei einer mittleren Temperatur von 30° bis 40° C gesäet und ergab keinen Brandbefall. Der andere bei 25° C in Poona gesäete Teil wurde zu 65% angesteckt.

Der Brand des Weizens (*Tilletia tritici* und *T. laevis*) ist in Indien von ähnlichen Faktoren beeinflusst. Er kommt in Kashmir und längs der Punjab-Vorgebirgshügel ständig vor, seltener und nicht gefährlich in dessen Talgebieten und ist weiter nach Süden und Osten zu unbekannt. Das Optimum für die Keimung der Brandsporen liegt ziemlich tief (zwischen 12° C und 18° C). Ueber 25° C kommt sie kaum je zustande und gerade dies ist die Temperatur, die gewöhnlich bei der Aussaat in Zentralindien oder dessen östlichen Gebieten herrscht, während sie in Nordwestindien zur Saatzeit tiefer liegt. Dort können die Sporen schon keimen und die jungen Pflänzchen befallen, süd- und ostwärts dagegen nicht mehr. HUNGERFORD (15) fand, dass in Idaho der stärkste Befall bei einer Temperatur von 0-12° C verbunden mit ziemlich hoher Bodenfeuchtigkeit eintrat.

Der Flugbrand des Maises (*Ustilago Zeae*) ist in Indien ähnlich verbreitet wie der Steinbrand, doch sind seine Beziehungen zur Temperatur noch nicht untersucht worden. In Amerika (16) hat man jedoch gefunden, dass das Maximum seiner Sporenkeimung und Entwicklung zwischen 30° und 38° C liegt, sodass zu erwarten ist, dass dessen Ausbreitung bei darüber liegenden Temperaturen zur Zeit der Aussaat in gleicher Weise als beim Brand der Sorghumhirse beschränkt sein wird; auch lässt seine Verbreitung in Indien diese Vermutung zu.

So ist also eine Gruppe der vier bedeutendsten Pflanzenkrankheiten in Indien (Kartoffelfäule, Brand der Hirse, Steinbrand des Weizens und Flugbrand des Maises) in ihrer gewöhnlichen Verbreitung streng durch die Temperaturbeziehungen von Wirtspflanze und Parasit bedingt, wobei der Einfluss auf den Parasit noch mehr ins Gewicht fällt.

In den Vereinigten Staaten ist in den letzten Jahren viel Tatsachenmaterial über die Temperatur- und Feuchtigkeitsbeziehungen bestimmter Krankheiten gesammelt worden. Nur ein paar der hervortretendsten Fälle sollen hier Erwähnung finden, in denen ein

Zusammenhang mit den durch die Gebiete bedingten klimatischen Bedingungen festgestellt worden ist.

Die an den Keimpflanzen des Maises und Weizens durch *Gibberella saubinetti* hervorgerufene Krankheit ist in den Vereinigten Staaten stark verbreitet, besonders in den westlichen Zentralgebieten. Es wurde bemerkt, dass der südliche wie auch der nördliche Teil des Korngebietes von der Krankheit nicht befallen wurde. Die Beziehungen der Krankheit zur Temperatur sind erst jüngst ausgearbeitet worden (17). Man baute Mais und Weizen auf Böden, die man ständig auf je 18 verschiedenen Temperaturstufen von 8° C bis 36° C hielt und der Ansteckung aussetzte. Es ergab sich, dass die für den Weizenbefall günstigste Bodentemperatur zwischen 12° und 28° C lag und ein Optimum von etwa 24° C aufwies. Mais, auf der anderen Seite, wurde bei 8-20° C angesteckt, u. zw. am stärksten bei 12-16° C. Der Befall scheint von der Wirtspflanze, nicht vom Pilz abzuhängen (18). Aehnlich den meisten *Fusarium*-Arten des Bodens zeigt der Pilz ein verhältnismässig hohes Entwickelungsoptimum (24-28° C). Man fand, dass es lange dauert, bis sich die Zellwände der Keimpflanzen im Mais bei tieferen und im Weizen bei höheren Temperaturen verstärken, sodass sie in dem Zustand vom Pilz leicht angegriffen werden können. Die Verstärkung der Zellwände geht beim Mais in höherer und beim Weizen in niedrigerer Temperatur rasch vor sich, wobei sie dann dem Eindringen des Pilzes Widerstand leisten können.

Für eine Menge Bodenparasiten sind die Temperaturbedingungen bearbeitet worden. Vier die Kartoffel schädigenden *Fusarium*-Arten weisen gemeinsam mit *Verticillium alboatrum* ein Entwickelungsoptimum von etwa 25° C auf, zwei *Fusarium*-Arten dagegen von 30° C. Die beiden letzteren *F. oxysporum* und *F. radicicola* (19), richten vor allem in den südlich gelegenen Kartoffelanbaugebieten der Vereinigten Staaten Schäden an. Der Parasit der Kohlfäule, *Fusarium conglutinans*, braucht für seine krankheitsverursachende Entwickelung ziemlich hohe Temperaturen. Bei einer Bodentemperatur zwischen 22° und 24° C wurden 97 von 104 Pflanzen angegriffen, zwischen 10° und 16° C 1 unter 88 (20). *Fusarium Lini* befällt den Flachs besonders zwischen 20° und 30° C; ähnlich wie bei letzter Krankheit ist seine Verteilung in Amerika eine beschränkte (21). Der *Fusarium* des Tabaks *F. oxysporium* var. *Nicotiana* tritt bei ähnlichen Temperaturen auf, deren Optimum für die Ansteckung bei 28° bis 31° C liegt, während geringer Befall um 15° C oder 34° C liegt. Dadurch erklärt sich

die Harmlosigkeit seines Auftretens auf bestimmten Flächen oder auf anderen in besonders heissen Jahrgängen.

All diese Fälle sind mit noch vielen anderen für die Temperaturverhältnisse von Wirtspflanze und Parasit nur im Untergrund bestimmt worden. Neuerdings ist man jedoch darauf gekommen, dass auch die Lufttemperatur, der die Pflanzenteile in der Krume ausgesetzt sind, von Bedeutung sein könne. Bei der Fusariumfäule der Tomaten (*F. Lycopersici*) (23) ist auch diese festgestellt worden. Bei der Geschwürbildung (sore-shin) der Baumwollpflanzen gleicht die Entwickelungskurve des Parasits sehr jener der Wirtspflanze, indem beide ihr Optimum um 28° C haben. Wird nun die Temperatur ständig auf 27-33° C gehalten, so bricht die Krankheit bei einer entsprechenden Bodentemperatur von etwa 27° C stark aus, nicht so bei 17° oder 33° C. Bei 17° Lufttemperatur zeigt sich kein Krankheitssymptom, einerlei wie die Temperatur des Bodens liegen mag, noch wenn diese unter 20° oder über 34° steht, einerlei, was dabei die Lufttemperatur macht, obgleich sowohl Wirtspflanze als Parasit bei jenen Temperaturen gedeihen können. Daher wird die Krankheit in Amerika vor allem in den Südstaaten und in England während der wärmsten Jahreszeit angetroffen.

Bei Zwiebelbrand (*Urocystis Cepula*) liegt der Fall ähnlich (24). Je nach der Boden- und Lufttemperatur können angesteckte Zwiebel zwischen 100 % und 0 % Befall aufweisen; dieser hängt dabei hauptsächlich von der Wirtspflanze ab. Die Keimung des Saatgutes kann zwischen 10° und 25° C Bodentemperatur erfolgen; sie ist jedoch bei 27° gering. Bei einer gleichzeitigen Boden- und Lufttemperatur von 25° C entgeht ein grosser Teil der Pflanzen durch die rasche Entwickelung dem Befall, bei einer Lufttemperatur von 30°-33° C kann er jedoch um sich greifen, wenn die Bodentemperatur unter 25° C gehalten wird. Bei Pflanzungen im Freien wurde der Befall schon bei einer Bodentemperatur von 29° vollständig unterdrückt, mit abnehmender Temperatur stieg er gleichförmig wieder an. Diese Tatsachen erklären die geographische Verbreitung der Krankheit in den Vereinigten Staaten; sie ist dort auf weiten Flächen nicht vorhanden, auf denen die Zwiebeln während heisser Bodentemperaturen gesetzt worden waren.

Selbst die Mosaikkrankheit zeigt ihre deutlichen Temperaturbeziehungen. Für ihr Auftreten auf Gurken sind hohe Boden- und Lufttemperaturen erforderlich; diese sind von DOOLITTLE (25) ausführlich untersucht worden. JOHNSON (26) hat ähnliche Angaben über

den Einfluss der Lufttemperaturen auf verschiedene Mosaikkrankheiten gemacht.

Die Feuchtigkeitsbeziehungen sind in den Vereinigten Staaten nicht für soviele Fälle bearbeitet worden.

Bei einem Vergleich zwischen dem Kronenrost des Hafers (*Puccinia coronifera*) und dem Getreideschimmel (*Erysiphe graminis*) konnte FROMME (27) durch Kronenrost keine Ansteckung bei einer relativen Luftfeuchtigkeit von unter 80 % hervorrufen, bei einer von 93 % nur zu 6 %; bei einem Feuchtigkeitsgrad von 100 % trat alsdann die Ansteckung ein. Schimmelbefall erhielt er dagegen bei 75 %-80 % Feuchtigkeit.

LAURITZIN (28) hat entdeckt, dass das Auftreten des Schwarzrostes (*P. graminis*) auf Weizen noch mehr begrenzt ist, indem es bei 92 % Feuchtigkeit nicht stattfindet und nur zwischen 95 %-100 % Erfolg hat. Das gleiche hat sich auch für *Colletotrichum Lindemuthianum* auf Bohnen ergeben und in sehr ähnlichen Schranken (91 %-100 %) auch für *Ascochyta Fagopyrum*.

Mit nur geringen Ausnahmen, wie bei der Kartoffelfäule (bei der ja die Temperatur eine grosse Rolle spielt) und bei der Schwarzfäule des Weines ist bisher wenig über die Feuchtigkeitsbeziehungen der Krankheiten gearbeitet worden. Der Grund hierfür liegt zum Teil an dem grösseren Aufwanderfordernis für genaue Untersuchungen, obwohl ziemlich feststeht, dass der Einfluss der Feuchtigkeit auf die Verteilung der Krankheit in verschiedenen Klimagebieten (ebenso wie auf die Stärke des Auftretens innerhalb eines Gebietes) ein ganz bedeutender ist.

Dennoch sind einige wenige genauere Arbeiten über die Feuchtigkeitsbeziehungen von Wirtspflanze und Parasit ausgeführt worden; diese schon zeigen zur Genüge, dass zielbewusste Forschungen in dieser Richtung hochbedeutsame Ergebnisse fördern müssten. So hat PANTANELLI (29) gezeigt, dass der *Plasmopara*-Befall des Weinlaubes von dem Zustände der Spaltöffnungen abhängig ist und nur dann auftreten kann, wenn diese sehr weit geöffnet sind. Junge Blätter sind infolge ihrer engen Spaltöffnungen immun. Auch bei den älteren Blättern tritt keine Ansteckung ein, wenn die Bodenfeuchtigkeit 15 % nicht überschreitet, es müsste denn die Luftfeuchtigkeit mehr als 80 % betragen. Bei über 20 % Bodenfeuchtigkeit öffnen sich die Spaltöffnungen stark, sodass sie bei gleichzeitiger Lufttemperatur von 40 % der Ansteckung Eingang gewähren.

Bisher habe ich mich auf die Wirkung einzelner Faktoren auf

besondere Krankheitsfälle beschränkt. Es würde hier zu weit führen, wollte man die Gesamtheit der klimatischen Einflüsse auf die allgemeinen Eigenschaften der Parasitenflora einer Gegend z. B. auf das verhältnismässig geringe Auftreten der Erysiphaceaen in den Tropen untersuchen. Ein oder zwei Fälle mögen jedoch erwähnt sein, um einen Einblick in die Bedeutung umfassender Forschung auf dem Gebiet zu geben.

In Tripolis hat TROTTER (30) Beobachtungen über die Pilzflora angestellt. Es herrscht dort ein heisses trockenes Klima; selten fallen Niederschläge und dann nur an den Küstenstrichen zwischen Oktober und Februar, im Binnenlande fast nie.

Unter den 83 beobachteten Basidiomyceten in weiterem Sinn finden sich 62 parasitäre Rost- und Brandpilze und von der gesamten Pilzflora sind nahezu 70 % Parasiten. TROTTER vermutet, dass das Wasserbedürfnis der Pilze dieses Missverhältnis zwischen harmlosen Pilzen und Parasiten hervorgerufen habe, da letztere ihr Wasserbedürfnis aus der Wirtspflanze befriedigen können und insofern von klimatischen Einflüssen unabhängig sind. Des weiteren sind kurzlebige Arten vorherrschend, besonders die Peuteromyceten. Unter den Rostpilzen weisen eine ungewöhnliche Anzahl kurze Kreisformen auf, ebenso wie die alpinen Rostpilze in der Schweiz; der Grund hierfür ist in der kurzen Entwickelungszeit zu suchen, die ihnen vom Klima gegönnt wird.

Aehnlich interessante Angaben wären von ökologischen Studien der Parasitenflora aus verschiedenen Gebieten zu erwarten, doch sind sie bisher noch äusserst spärlich betrieben worden. DUFRENOY (8) untersuchte von diesem Gesichtspunkte aus das Talgebiet von Barèges im Norden der Pyrenäen. Er untersuchte den Einfluss der Höhe auf die Parasiten bestimmter Pflanzen, die vom Saume der Hänge in den Tälern bis zu 2 000 m hoch gedeihen. Er konnte dreierlei Gruppen unterscheiden. In der einen traten die Parasiten in jeder Höhenlage gleichstark auf (die meisten Ascomyceten), in der anderen stärker in den tieferen als in den höheren Abschnitten (einige Brandarten, Kartoffelparasiten), und in der dritten waren sie hauptsächlich in den höher gelegenen Gebieten zu finden (Eichenmehltau und einige Brandarten). Er schloss, dass die Strahlung bei der verschiedenen Höhenverteilung der Parasiten den bedeutendsten Einfluss ausübe. Die Temperatur war insofern von Bedeutung, als sie die Höhe des Schadens und sein zeitliches Auftreten bedingte, doch übte sie nur geringe Wirkung auf das Vorhanden- oder Nicht-

vorhandensein des Parasiten aus. Die Wirkung des Schattens wurde ausführlicher untersucht, wobei sich die Intensität und Stärke der Sonnenbestrahlung als der bestimmende Faktor für die Entwickelung der Blattparasiten ergab.

Das praktische Interesse an der Bedeutung für das Studium der Beziehungen zwischen den meteorologischen Bedingungen und den Pflanzenkrankheiten braucht kaum besonderer Anregung. In Frankreich (31) ist seit einiger Zeit eine meteorologische Nachrichtenstelle für Weinkrankheiten in Tatigkeit, die von 60 Unterstationen aus bedient wird. Sie unterrichtet über deren voraussichtliches Eintreffen und über rechtzeitig zu treffende Vorkehrungen, wie Spritzen und Schwefelung. Die Voraussagen grunden sich auf die Kenntnisse der Bedingungen, die die Keimung, die Ansteckung, das Incubationsstadium in den Geweben und endlich das aussere sichtbare Auftreten der Parasiten beeinflussen; sie werden uberdies durch Beobachtungen auf den Feldern der Station nachgeprüft. Es ist auf diese Weise möglich, 8 Tage vorher das sichtbare Auftreten der Sporen zu verkünden, sodass noch reichlich Zeit für Schutzvorkehrungen verbleibt. In Italien arbeitet von Turin aus (32) eine ähnliche Stelle.

In Deutschland hat die Biologische Reichsanstalt in Berlin-Dahlem ein Laboratorium für Meteorologie und Phanologie errichtet, das durch ein Rundfragesystem im ganzen Land (33) mit der Sammlung von Angaben begonnen hat. Eine begrenzte Zahl kennzeichnender Krankheiten soll ausgelesen werden; man will versuchen, ihr Auftreten und ihre Verteilung in verschiedenen Gebieten mit den Isothermen und mit anderen meteorologischen Daten, sowie mit den jährlichen Schwankungen der Jahreszeiten in Uebereinstimmung zu bringen. Auch wird eine Sammlung von Leitpflanzen durchgeführt, die einen parallelen Verlauf zu den Beziehungen zwischen Jahreszeit und Parasitenauftreten aufzuweisen haben.

Das United States Weather Bureau (die Wetterwarte der Vereinigten Staaten) führt zurzeit eine ausgedehnte Untersuchung über die Bestimmung der Wirkung laufender meteorologischer Faktoren auf das Pflanzenwachstum aus. Als Teilarbeit daraus werden von verschiedenen Anbauzentren systematische, meteorologische und plänologische Aufzeichnungen gesammelt, die den Zweck haben, die kritischen Punkte in der Vegetationsdauer der Pflanzen und in der Entwicklung der schädlichen Insekten oder Pilze zu bestimmen.

Obwohl die Angaben bis jetzt noch nicht genügen um genauere Schlüsse auf den Befall durch Krankheiten zu erlauben, so hat man doch schon einige bemerkenswerte Zusammenhänge feststellen können. So macht die Kartoffelfäule bei einer täglichen Durchschnittstemperatur von 72° F ihre stärkste Entwickelung durch; auch wir haben schon oben erwähnt, dass das Temperaturoptimum für die Entwicklung des Parasiten in der Wirtspflanze etwa 24° C oder 75° C beträgt.

Die Aufgabe dieser Mitteilung liegt jedoch mehr in dem Hinweis auf die Bedeutung des Heraustretens an solche Arbeit und weiterhin auf die Wichtigkeit, zuerst die Temperatur- und Feuchtigkeitsbeziehungen zwischen Parasit und Wirtspflanze einzeln und gemeinsam genau zu bestimmen, und erst danach den Zusammenhang zwischen den gefundenen Tatsachen und den meteorologischen Angaben zu suchen.

Die Mitarbeit der meteorologischen Sachverständigen sollte man erst in dem Augenblick aufsuchen, in dem man die Ergebnisse der genauen Untersuchungen praktisch zu verwerten sucht. Es ist zweifelhaft, ob die meteorologischen Berichte, wie sie heutzutage herausgegeben werden, allein genügen. In Indien tun sie es nicht, weder inbezug auf die Temperatur noch auf die Feuchtigkeit. Selbst da, wo die Aufschreibungen dreimal am Tage erfolgen, ist eine Gleichung zur Verbesserung des wahrscheinlichen Fehlers nötig. Mehrere solcher Gleichungen sind für die Durchschnittstemperaturen ausgearbeitet worden, doch schwankt ihre Anwendungsmöglichkeit in den verschiedenen Gegenden. Für die hier angeführten Zwecke sind ausserdem nicht nur die täglichen Durchschnittstemperaturen, sondern auch die Dauer der täglichen Extreme von höchster Bedeutung, kann doch ein geringes Minimum, wenn es nur lang genug zu wirken vermag, vielleicht für einen Parasitenbefall genügen, was aus den täglichen Durchschnittszahlen, wenn sie durch einen heissen Tag ausgeglichen werden, nicht herauszulesen ist.

In diesem kurzen Ueberblick sind viele sehr bedeutende Gesichtspunkte, wie z. B. die Frage der Ueberwinterung der Getreidebrandarten ausgelassen worden. Es wurden nur Beispiele über die verschiedenen Einflüsse der Umgebungsfaktoren auf die Pflanzenkrankheiten gegeben. Feldbeobachtungen haben für das Auftreten von Krankheiten in den verschiedenen Jahreszeiten und Gebieten zweifellos einen gewissen Wert, doch war es Absicht dieser Arbeit, zu zeigen, dass ein-

deutige Schlüsse aus solchen Beobachtungen nicht gezogen werden können, es sei denn sie werden durch die genaueren Methoden wissenschaftlicher Untersuchungen im Laboratorium überprüft und gedeutet.

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THE WORK OF THE IMPERIAL BUREAU OF MYCOLOGY

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THE WORK OF THE IMPERIAL BUREAU OF MYCOLOGY

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THE serious study of the depredations caused to cultivated crops by parasitic fungi and bacteria dates from comparatively recent times, and it was not until after the beginning of the present century that any organized attempt was made to ascertain the cause and seek methods of prevention of crop diseases in the tropical and sub-tropical parts of the Empire. As agricultural departments were established in the various dependencies, officers were appointed who were charged with the duty of investigating the diseases of plants and devising means for their prevention or control. These officers are usually termed " mycologists," though the names " plant pathologist " or " phytopathologist," used in other countries, more aptly describe their functions. There are not far short of 100 such experts at present at work in the overseas parts of the Empire, all, with very few exceptions, attached to Government Departments of Agriculture.

In some cases these workers enjoy facilities not inferior to those available in the best equipped laboratories and research stations in England. In others, and this applies particularly to the tropical areas, certain facilities are poor. The chief lacks in such cases are a good library and access to authentic specimens of the parasites known to cause disease in plants. There is nothing more discouraging than to find that work which has cost much time and labour has already been done somewhere else, or that a supposed new parasite has previously been studied and its ways are known. Even in the best equipped centres it is difficult to keep pace with the mass of research and practical work that is going on in many parts of the world, and to see and find time to read all the publications (many in obscure journals or difficult languages) that have a bearing on one's work.

In the past, assistance in such matters was provided by the Royal Botanic Gardens, Kew, and the British Museum of Natural History at South Kensington. Germany had carried the systematic study of the subject beyond the point reached by any other country, and German assistance was almost indispensable to the overseas worker. With the outbreak of war the inadequacy of these arrange-

ments became painfully evident, and both in England and in the dependencies means were sought to satisfy the requirements of the scattered band of mycologists throughout the Empire. The Imperial Bureau of Mycology was the result.

The Bureau was founded in consequence of a resolution of the Imperial War Conference passed on July 8, 1918. The subject was brought to the notice of that body by the Colonial Office, which, impressed by the success which had attended the efforts of the Imperial Bureau of Entomology, founded five years earlier to meet a similar situation in regard to insect pests, thought that the time had come to establish a like organization to deal with the other great class of destructive agencies in agriculture—namely, the diseases of plants caused by fungi. A number of the overseas Governments expressed their readiness to contribute to the support of the Bureau, and work was eventually started in September, 1920. The affairs of the Bureau are managed by an honorary committee appointed by the Secretary of State for the Colonies, with Earl Buxton, G.C.M.G., as Chairman. Its finances are on a temporary footing, and negotiations are in progress to extend them for a period of five years from April 1, 1925.

The work of the Bureau falls naturally into two main divisions: it serves as a clearing-house for information on all aspects of plant pathology other than those concerned with insects and the like, and it examines and reports on specimens sent in from all parts of the Empire.

As a centre for information the Bureau endeavours to keep in touch with the work on the diseases of plants going on throughout the world. A staff of linguists is engaged in reading all the literature on the subject published in any part of the world to which access can be had, and in making abstracts of all original papers. These abstracts are printed monthly in a journal published by the Bureau, the *Review of Applied Mycology*. They are given in sufficient detail to enable workers in many cases to dispense with reference to the original paper, and are issued sufficiently promptly to keep up a survey of current activities in the whole field of plant pathology. The monthly numbers at present contain from 100 to 120 abstracts, and the volume for 1924 runs to 752 pages, exclusive of the index. The latter is in great detail, as the value of an abstracting journal in after years depends on the readiness with which references to particular matters may be looked up. No attempt is made to secure a satisfactory balance-sheet in regard to the *Review*: to do so would be impossible without charging a prohibitive price per

copy, as the circulation is practically limited to the comparatively few who are directly interested in the subject. Hence an appreciable proportion of the Government grants is applied to the production of the *Review*, and copies are supplied free of charge to all the contributing Governments for the needs of their departments. At the recent Imperial Mycological Conference, held in London last July, a resolution was passed expressing appreciation of the service rendered by the *Review* to all workers on the subject, especially those in the overseas parts of the Empire. It is probable that no branch of the work of the Bureau has been more widely approved than the publication of this journal, and its reception has been scarcely less cordial in America and on the Continent than within the Empire.

Another aspect of the information service is the furnishing to enquirers of references regarding previous work on a problem. In order to deal with such enquiries the Bureau aims at keeping up a comprehensive card index to the literature on plant diseases, and, in addition, maintains a large lending library of original papers in pamphlet form available for issue on demand. This library numbers over 6,000 items, cross-indexed by subject and author, and is increasingly being made use of by overseas mycologists. Extracts and translations from publications not easily available are also asked for at times, and requests for information of one kind or another are received in considerable numbers, almost by every mail. Recently a demand has arisen for copies of sections of the subject card index to be made available for some of the larger departments, and these are being issued at cost price.

The examination of, and reporting on, specimens sent from overseas is the second main activity of the Bureau. Progress in this branch of mycology is less advanced than in regard to insects. The fungus flora of a great part of the British tropical and subtropical dependencies is not well known; an enormous amount of work remains to be done before an adequate account could be given of, say, the fungi of tropical Africa. Plant disease surveys have been undertaken in some areas, but these are, as a rule, confined to recording the diseases which cause notable damage to important crops. Unfortunately, a parasite that may be inconspicuous and relatively harmless in one area may cause a major disease in another. Many years ago the writer collected an insignificant leaf-spotting fungus on cotton in Kashmir which proved to be new to science. Nothing more was heard of this until, in 1920, a severe outbreak of a new disease of cotton was reported from Arkansas, and on investigation was found to be due to the same fungus. How it got from Kashmir

to Arkansas (or *vice versa*), and what countries in between it affects, are not known, but it is evident that every record of its distribution would be of importance.

Within the limits of the capacity of its small staff the Bureau tries to aid in gaining a more detailed knowledge of the fungi of the colonies. In some, such as Ceylon, little or no outside help is required, for such knowledge is already far advanced. In others, intensive collection is much needed, and it will be many years before the survey is completed. The number of systematic specialists available for the study of these collections in England is small, and progress is likely to be slow even with the aid of foreign workers.

In addition to this general systematic work a large amount of time is devoted to the examination of individual specimens, often of obscure diseases or of parasites whose identification is difficult. In regard to this part of the work the position of the Bureau is somewhat like that of a consulting physician, except that it does not aim at dealing with all such enquiries itself, but passes many of them on to other specialists. In every case the idea is to get the best advice available, whether in England or abroad. The organization of the information side of the work is called into service here, since it is through the latter that the Bureau keeps in touch with active workers in various parts of the world and is usually in a position to know at any given moment who are working on any disease or group of fungi that the specimen may belong to.

A couple of cases illustrating this branch of the work can be taken from enquiries regarding cotton diseases that have been dealt with during the past year or two. The outbreak of black arm disease, caused by *Bacterium malvacearum*, in the Sudan, is one of the serious problems which the Sudan Plantations Syndicate has to face. The Bureau has been in close touch throughout with Mr. R. E. Massey, the Sudan Government Botanist, in whose hands the investigation of this problem is placed. The first isolations of the parasite were sent to the Bureau to confirm its identity with *B. malvacearum*, and new cultures made from these were transmitted to Dr. Erwin Smith at Washington, the original describer of the parasite. He tested it fully, and reported that it was a variety of this organism, which was known also in the United States, but had not been described in detail. It is probable that without such an authoritative opinion there would still be doubt whether the disease was really black arm. Furthermore, the search for a satisfactory method of disinfecting the seed has been aided by the Bureau putting Mr. Massey in touch with institutions and firms most likely to be of

assistance to him. The recent promising trials of izal against the same disease in the West Indies were arranged entirely through the Bureau. The prevalence of black arm in Nigeria has just been reported to the Bureau, which is thus in a position to put the local mycologist in direct touch with practically all that is known about it elsewhere.

A second interesting case is concerned with an obscure fungus which has been known to attack cotton and other plants in India for a number of years. More recently Mr. Briton-Jones, the late Mycologist to the Ministry of Agriculture in Egypt, found a similar fungus injuring cotton in that country. Dr. Shaw of Pusa, India, suggested that the Bureau should compare the parasite with a fungus, *Sclerotium bataticola*, which attacks sweet potatoes in the United States. Cultures of the Indian, Egyptian, and American fungi were grown side by side at the Bureau and proved to be identical. The curious thing is that the fungus has not been reported on cotton in America, while in the other countries it has not been found on sweet potatoes. Quite recently all three strains have been tested on sweet potatoes in Uganda, and all were found to rot the tubers just as described in America. In Uganda the same fungus infects beans, and will doubtless be found on cotton too, while from Nigeria it has recently been received on cotton and ground-nuts.

It can easily be realized that a single enquiry of the nature of those mentioned above may occupy several months and involve, in such cases as the last, a good deal of laboratory research. Apart from this kind of work, however, the Bureau does no original research, since it has always maintained the view that research into the cause and control of plant diseases must be done on the spot.

While the Bureau, for obvious reasons, works as a rule through the local departments of agriculture, its services are freely available to non-official mycologists. These are unfortunately few as yet, but the example set by the Indian Tea Association, the Rubber Growers' Association, and a few large corporations such as the Colonial Sugar Refining Company in Australia, in maintaining their own research staffs (including mycologists), is worthy of wider imitation. The existence of the central organization outlined above will undoubtedly make such a policy easier to adopt, and will smooth the path of the individual workers in small, isolated scientific centres, such as one may hope will be founded in increasing numbers by the plantation industries in the overseas parts of the Empire.

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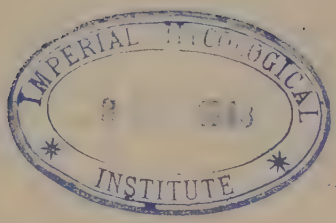

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THE WILT DISEASES OF COTTON AND SESAMUM IN INDIA

BY

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The occurrence of wilt diseases of cotton and sesamum in India has been frequently recorded, and the purpose of the present paper is to describe some unpublished work on them carried out in 1912, and to discuss briefly the relationships of some Indian wilt-producing fungi.

Cotton wilt.

Fresh material of cotton wilt was selected by the writer from a field of Jari cotton at Matargaon village in the Berars, India, on 2nd October, 1912. There was a large patch of wilted plants in this field, and the specimens were taken from five plants in an early stage of the disease, each of which had still some of the leaves green.

Isolations were attempted on the same day from each of the plants by washing a piece of infected tap root in several changes of sterile water, dipping in absolute alcohol, flaming off the alcohol, and dropping into prepared tubes of medium or into sterile Petri dishes. Microscopic examination of adjacent sections of the root showed in each case the presence in the vessels of hyphæ resembling those found in the wilt disease of pigeon pea (*Fusarium udum*). Growth was obtained on the 5th from three of the plants thus treated, the pieces of root from the other two remaining sterile. Subcultures were made on nutrient agar from the roots of these three plants on the same day. The fungus was similar microscopically on all three and was found in two cases to be bearing micro- and small macroconidia of a *Fusarium*, while the third had no spores on the young piece of the mycelium examined. On returning to Pusa on 17th October, good growth was found in the subcultures from two of the plants, that from the third being sterile. The fungus was similar in both cases and was a *Fusarium* with numerous microconidia and a few macroconidia. Later on large quantities of chlamydospores developed in the tubes; in cultures these were collected into stromatic, nodular masses. The inoculations described below were from one only of these isolations.

On 1st August, 1913, four culture pots that had been sterilized after filling by heating in the pressure boiler of the Pusa pot culture laboratory were inoculated by adding to the soil a suspension in distilled water of this fungus from a pure culture

Fig. 1. A wilted and healthy cotton plant growing in inoculated soil.

Fig. 2. A wilted and four healthy cotton plants growing in inoculated soil.

Fig. 3. Inoculated and control pots of sesamum, former attacked by wilt.

six days old. On 2nd September they were sown with Bani cotton seed sterilized by soaking in 0·25 per cent. formalin for three hours, a fresh suspension of the fungus from a culture of the same date as the last being added around each seed. Two other pots similarly treated, except that they were not inoculated, served as controls. Six more pots that had been sterilized after filling on 29th August were sown with the same seed on 2nd September, four being inoculated from the culture last mentioned by adding a suspension around the seed, and two left uninoculated. Thus four of the pots had received a double inoculation, one to the soil a month before sowing and a second with the seed, while four had received the latter only, and there were four controls. Germination was poor and all the pots were resown with sterilized seed of the same variety on 17th September. The first case of wilt in the inoculated pots was observed on 23rd October, and from that time until 18th December, when observations ceased, cases continued to occur sporadically in five of the inoculated pots. No cases occurred in the other three inoculated or in the control pots, one of which was noted as having 11 healthy plants, 6 to 8 inches high, on 29th November. The conditions in four of the pots are described in detail below.

In a pot containing three plants, one of these plants, about 6 inches high and with about 6 leaves, began to wilt on 10th November and it died in the next few days. A second began to droop on 17th November and was dead two days later. It was removed and sectioned. The lateral roots were little altered except for some brown stains, especially marked near the junction with the tap-root. The latter was not outwardly affected. The collar was shrunken. Hyphæ and chlamydospores were present in the vessels about 1 inch above soil level, but were not numerous. Mycelium was also found in the tissues 1½ inches below the soil, chiefly in the vessels and in the centre of the central cylinder. At 3½ inches below the soil, the tap and lateral roots were free from fungus. A microconidium was found in a vessel near the soil level. The fungus was recovered in a manner similar to that followed in the original isolations, and pure cultures on nutrient agar secured. It resembled the fungus used in the inoculations and yielded micro- and macroconidia and chlamydospores.

On 16th November one plant in a second inoculated pot, in which there were also a large plant (9 inches high and with about 8 leaves) and a smaller seedling, both quite healthy, began to wilt, and wilting was complete on the 19th when the small seedling was pulled out and the pot photographed (Plate XIII, fig. 1). The other two plants were then removed and sectioned. No trace of fungus infection was found in the tap-root, collar, or stem of the healthy plant, while in the other there was strong infection extending from the stem above ground to the fine part of the tap-root about three inches below. The vessels were in places almost blocked with a dense, matted growth of hyphæ, and numerous microconidia were found in them. The fungus was recovered as before and pure cultures bearing micro- and macroconidia and chlamydospores were obtained.

On 23rd November a strong plant about 8 inches high, in a third inoculated pot in which there were 4 other quite healthy plants, began to droop. The pot was photographed on the 26th (Plate XIII, fig. 2), and the next day the wilted plant (which was still quite green but flaccid and exactly as if it was wilting from lack of water) was removed and examined. Hyphæ were found in the vessels an inch above soil level and in much greater quantity three inches lower down. The lateral roots in this, as in most of the other cases, were externally quite unaltered. At 3½ inches below the soil hyphæ were rare in the tap-root. On 1st December a second plant in this pot began to wilt and by the 5th it was quite dead, while on 17th December the biggest plant in the pot wilted and all its leaves were drooping on the next day, when observation ceased, the two remaining plants being still quite healthy.

On 10th November the only plant in another pot began to wilt. It was about 6 inches high and had 6 leaves. Three days later it was removed and sectioned. Hyphæ with some microconidia were present in the vessels in the neighbourhood of the collar, but the lateral roots were for the most part white and sound. It appeared to be a typical case of cotton wilt. The pot was resown on 17th November and three seedlings came up. One of these wilted on 8th and another on 16th December, one being still quite healthy two days later when observation ceased.

The first three cases described above belonged to the batch of pots that had received a double inoculation, while the fourth had only received one. Two plants out of seven wilted in another pot of this latter batch, but no deaths that could be definitely attributed to wilt occurred in one pot of the first batch and two of the second. Thus the percentage of deaths from wilt in the inoculated pots was low and the incidence of the disease was irregular, some plants escaping altogether while others succumbed, though all must be considered to have been exposed to the same intensity of infection. Nevertheless, those that became infected died in the manner characteristic of the *Fusarium* wilts, the examination of a large number of wilted plants in the field supported this conclusion, and the capricious results of the inoculations would appear to be due to the action of some factor which aids or hinders infection by the fungus. That this factor cannot be a soil one is highly probable from the fact that the one batch of soil was used for each of the two series, while even in the same pot some plants wilted with heavy infection though others survived and were free from internal hyphæ.

The experiments above described do not solve the problem of cotton wilt in Central and Western India. They would not have been published as they stand were it not that circumstances render it unlikely that the writer will be able to do any further work on the disease. It is believed that they show that the disease is caused by a *Fusarium* which is capable of pathogenic action under certain conditions not yet elucidated, and that these conditions are probably not connected with the composition of the soil, as has been suggested by J. F. Dastur.[1]

[1] *Agri. Jour. of India*, Vol. XIX, pp. 251-260, 1924.

Sesamum wilt.

Isolations were made from wilted seedlings of sesamum (*Sesamum indicum*) about 6 weeks old and 4 to 6 inches high at the Hoshangabad experimental farm, Central Provinces, India, on 28th September, 1912. The isolations were made in the same way as those already described in the case of cotton wilt, and a pure culture on nutrient agar was brought back to Pusa and subcultured on 17th October. The fungus was a *Fusarium* with all types of spore from oval, cylindrical, or falcate, continuous microconidia to fusiform or slightly falcate, pluriseptate macroconidia. Chlamydospores were numerous, single or in chains, round or pear-shaped, colourless, and charged with distinct fat droplets.

On 1st August, 1913, a subculture of this fungus, six days old, was used to inoculate four pots of sterilized Maghai sesamum, the filled pots and the seed having been sterilized as in the cotton wilt work except that the seed was immersed in 0·25 per cent. formalin for only two hours. After sowing, but before covering, a suspension of the fungus in distilled water was poured over the seed. Two other pots similarly treated but without inoculation were kept close by as controls.

On 8th August a seedling in one of the inoculated pots collapsed at the collar, and next day cases of wilt were observed in all but the control pots. On the 11th the four inoculated pots had respectively 10, 7, 12, and 8 plants healthy, 1, 1, 0, and 3 dying and 5, 5, 6 and 7 dead, while the two controls had a number of healthy seedlings and only one dead. On the 14th there were 1, 3, 2 and 1 healthy, 0, 3, 0 and 0 dying, and 15, 7, 16 and 17 dead in the four inoculated pots, while in the controls three more plants had begun to wilt. By the 20th all the seedlings had died of wilt in the inoculated and in one of the control pots, which had 30 plants, while in the other control, with 23 plants, all were healthy. The deaths were similar throughout; brown stains showed at the ground level and the plant rapidly became flaccid and fell over. They were quite obviously due to virulent attack by the fungus, which was forming spores freely on the surface of the affected plants and had spread to one of the controls which was standing alongside the inoculated pots.

On 2nd September the five pots in which all the seedlings had wilted were resown with sterilized seed as before, a fresh inoculation being made by adding to water poured around the seed a suspension from a culture of the same date as that previously used. Six more freshly sterilized pots were also sown on the same day in the same manner as in the first experiment. Three of these were inoculated as before and three kept as controls. The latter were placed in an isolated position in another building at a distance from that in which the inoculated pots stood. Germination was very good and the first case of wilt was observed in one of the resown pots on 7th September. On the next day there were in the five resown pots 39, 28, 48, 35 and 35 seedlings respectively, of which 3, 0, 6, 2 and 1 were wilting; in the newly-sown inoculated pots there were 39, 26 and 33 seedlings all healthy, and in the controls a large number also all healthy. The first case of wilt in the newly-sown pots occurred on the 9th, and next day there were several cases in each of the three

inoculated pots of this batch. The photograph in Fig. 3 (Plate XIII) was taken on the 15th and shows the contrast between an inoculated pot (that which had 48 seedlings all of which but 3 or 4 were now wilted) and a control with 45 seedlings, all healthy. By 23rd September all were dead in the inoculated pots except in one of the freshly-sown ones in which 4 were still alive, while all were healthy in the controls.

Several apparently healthy seedlings were removed from one of the newly-sown inoculated pots on 16th September and examined. They showed red markings at the collar about the junction of root and stem. Hyphæ were present in the epidermis and cortex at this level but none had penetrated the endodermis. On young seedlings such as those exposed to infection in these experiments the parasite kills more in the manner of the damping-off fungi than in that characteristic of the vascular wilts. Its main development takes place in the parenchymatous tissues and it forms, under suitable conditions of moisture, a considerable growth of hyphæ and spores on the collapsed aerial parts of the shoot. On older plants, specimens of which have been examined from time to time, the disease is more like a typical wilt such as that caused by *Fusarium udum*, and in such cases entry is probably through the finer roots, extension upwards occurring through the vascular system.

Whether the disease is the same as that in Turkestan sesamum described by Jaczewski (*Annales Mycologici*, I, pp. 31-32, 1903) and attributed by him to *Neocosmospora vasinfecta* cannot at present be determined. Jaczewski's description is brief and purely morphological, so that it would be scarcely possible to do more than recognize his fungus as a *Fusarium* probably of the *Elegans* section of the genus were it not that, shortly after, Bessey gave fuller details of what was evidently the same fungus, which he isolated from wilted sesamum plants similarly received from Turkestan. His cultural studies (*Flora*, XCIII, pp. 301-334, 1904), joined to Jaczewski's morphological description, leave practically no room to doubt that the Turkestan sesamum wilt is caused by the same fungus as that in India. Although the latter author determined the parasite as *Neocosmospora vasinfecta*, and Bessey found great similarities in its cultural characters with those of that fungus, subsequent work both in India and the United States enables us to exclude the latter from consideration.

The study of a new series of cultures from wilted sesamum plants, which I owe to the kindness of Mr. J. F. Dastur, Mycologist to the Central Provinces Government, Nagpur, has not only enabled me to follow the colour changes described in such detail by Bessey, but has also brought out many points of similarity to other species of the same section of the genus. Comparison with *F. cubense*, the cause of the Panama disease of bananas, revealed no constant morphological or cultural differences. Furthermore, I have vainly endeavoured to find a true distinguishing character between the sesamum fungus and *F. udum*, described by me in 1910 as the cause of the pigeon pea wilt in India (*Mem. Dept. of Agri., India, Bot. Ser.*, II, 9). Small has recently studied in great detail what he considers to be *F. udum*, which he found attacking a number of different plants in Uganda (*Kew Bull.*, 1920, p.

321 ; 1922, p. 269 ; 1925, p. 118). Hence, though in India the strain of *F. udum* parasitic on pigeon pea seems to be restricted to that host, in Uganda the morphologically and culturally similar fungus is capable of attacking not only pigeon pea but a considerable number of other plants, and this strengthens the possibility that the sesamum parasite may be merely another strain of the same species.

Fusarium udum itself was named without prejudice to the question whether it had not been previously included amongst the named members of the genus. The chief diagnostic characters were the pionnotal type of sporulation, the flesh to salmon-pink colour on many media with absence of blue colours, and the tubercular stromata on potato and plantain. Subsequent isolations at Pusa, however, showed that the first of these characters was not constant, some strains giving a copious aerial mycelium on agar slants. Nodular sclerotia also are now known to be commonly produced by many members of the genus and occur in both *F. cubense* and the sesamum parasite, in which they are blue on potato but gradually turn pink if placed in lactic acid. Bessey's conclusion that the red and blue colours are only chemical modifications of the same pigment has been substantiated by subsequent investigators, and there seems every probability that the blue or violet colours developed in *F. cubense* and the sesamum fungus could also be produced by *F. udum* on suitable media. Small, indeed (*Kew Bull.*, 1922, p. 282), obtained a pale blue pigment in his strain in two cultures.

Recently, Hansford, confronted with the difficulty of distinguishing *F. cubense* in culture from many other strains of the *Elegans* section of the genus, has concluded that the present classification of the section is useless and that the conception of a species of *Fusarium* must be broadened (*Proc. 9th West Indian Agri. Conf.*, 1924, pp. 43-44, 1925). He considers that it is preferable to regard all the *Elegans* forms which he has encountered as strains of a single species. With this view, my own observations are in harmony, and I now regard *F. udum* and the two forms discussed in the present paper as strains of the same species. Furthermore, *F. cubense* is so similar to the sesamum fungus that it can scarcely be considered as a distinct species, either on morphological or cultural characters. It appears to possess strains differing in their selective parasitism, *Musa cavendishii* being immune from it in the West Indies but susceptible in the Canaries. Finally, through *F. cubense*, which both Brandes and Woollenweber have noted to be scarcely different from *F. vasinfectum*, one is led to include the latter in the group of closely allied strains. In so doing I have reversed my previous opinion (*Rept. Agri. Res. Inst. and College, Pusa, 1913-14*, p. 54, 1914) which was based on cultural differences between the American and Indian cotton wilt fungi, these differences being now regarded as too inconstant to be used as satisfactory criteria.

Thus the wilt-producing fungi attacking cotton, sesamum, and pigeon pea in India may, in the writer's opinion, best be considered to be strains of *Fusarium vasinfectum* Atkinson, which itself may be merely a strain of one of the earlier described species of the genus.

In many respects, Farrer conducted his work along lines which hold good to-day, and were definitely scientific. His parent types were selected with the definite ideal of combining desirable characters from each parent in an improved strain of the progeny. His selection and fixing was done on the single plant basis, which has been proved sound by Mendelian research. Modern genetic research has explained many phenomena which were inexplicable to Farrer, and has placed the fixing and selection of what might be termed intermediate characters on a more definite footing, but, in general, Farrer was working on sound lines. Striking exception was his habit of crossing unfixed cross-breds, even first generation of plants. This enormously reduced his chance of obtaining desired recombinations, as compared with the chance which would have followed crosses between parent types, fixed or pure, for the respective characters desired. Farrer's practice was, however, based on the theory prevalent in the nineteenth century that crossing simply resulted in a promiscuous "breaking of the type."

Progress in Australian wheat-breeding will most probably result from what might be called "character" breeding rather than from generalized crossing and selection. The weak points of our present standard varieties should be improved by crossing made with the object of introducing new characters such as resistance to flag-smut, rust, take-all, &c., the improvement of the yield of early maturing varieties, and the combination of yield with quality. To facilitate this, investigation work is required in regard to genetic and physiological problems of the genus *Triticum*. Such research is fundamental to further marked progress.

32. Practical Methods of Improving the Yield of Maize by Breeding and Selection.

By H. Wenholz, B.Sc.Agr., New South Wales Department of Agriculture.

[*Abstract.*]

Mass selection (as distinct from pedigree selection) is the usual mode of selection applied to maize by farmers in Australia, and if intelligently carried out is capable of effecting striking qualitative improvement, and of at least maintaining the yield of a variety of maize. The establishment of correlations of visible characters in the ear and plant with yield is not definitely established, and it has been found that farmers have somewhat too dogmatic views as to the importance of some characters such as deep grain, thin core, well filled tips, straight rows, and high shelling percentage, and their supposed relation to high yield. If less rigid selections for these characters be

practised, and more attention given to the selection (especially in the field) of sound, mature, plump, heavy seed of a variety of maize suited to local conditions, it is thought that some improvement may be made in yield as well as quality by mass selection. Especially is this the case where farmers procure seed of the same variety from different sources for getting "fresh blood" into their maize. Good results have followed from this course. Investigations are in progress at the experiment farms for producing a superior strain of maize suitable for introducing this "fresh blood" into farmers' seed, and these have already had considerable success as judged by the result of maize contests in the coastal districts. It is thought that this function of the experiment farm is more important than that of supplying large quantities of seed maize which can well be, and is being, well undertaken on a commercial scale by farmers. Stress is laid on the necessity for reaching farmers throughout the state with a simple method of maize improvement rather than confining investigations to involved technical methods which cannot be advised for the average farmer, even though they may be highly successful in the hands of the skilled breeder.

DISEASES OF CEREALS.

33. The Effect of Climate on Cereal Diseases.

By Dr. E. J. Butler, Director, Imperial Bureau of Mycology, London.

While in the earlier days the study of plant diseases resolved itself into little more than a study of the parasite, and while more recently greater prominence has been given to a consideration of the behaviour of the host plant when attacked, it is only within the last few years that it has been fully realized that disease is a complex interaction of host and parasite, and that this interaction is much affected by variations in the conditions of the environment.

No observant person who watches the incidence of plant diseases can avoid noticing the marked influence of one group of environmental factors, those included in what we rather vaguely term the weather, on the prevalence of many of these diseases. The powdery mildews (*Erysiphaceæ*), for instance, are often worse in dry years, whereas the downy mildews (*Peronosporaceæ*) are usually destructive in wet years.

The effect of these variations in the weather of any one area from year to year is naturally exceeded by that produced by the variations in climate between different countries, or different parts of countries, large enough to possess different climates (that is different *average* weather conditions), owing to the usually greater amplitude of the

variations in different climates than in the weather of any one climate. In this connexion, it should be borne in mind that man has succeeded, by conscious or unconscious effort, in extending the range of important cultivated plants into different climates whereas the parasites have not been subjected to the same influence. In many cases (e.g., in America and India) the range of such cereals as wheat, maize, and sorghum, is much greater than the range of certain of their parasites.

Weather and climate depend on the ordinary meteorological factors —temperature, humidity, radiation, wind, &c., and if we wish to understand the relationship between meteorology and diseases, we must attempt to evaluate the influence of each of these factors on the interaction between host and parasite. I believe that this influence is capable, for the most part, of exact evaluation, though the data are but scanty as yet. Temperature, humidity, and radiation are the chief factors about which we have some exact information.

The most difficult case is undoubtedly the study of the influence on disease of the less marked variations in any given climate from year to year. These weather changes are frequently not large enough to prevent the disease from appearing, but merely modify its amount. Variations between different climates may, on the other hand, entirely eliminate a disease.

In the greater part of India, wheat rust is present every year in probably every wheat-field. Whether an epidemic outbreak occurs or not appears to depend entirely on the weather conditions of the year. These conditions were analyzed by Mr. W. H. Moreland, of the Indian Civil Service, when Director of Agriculture in the United Provinces, for thirteen years in three selected districts. He concluded that the deciding factor was the humidity of the air in January and February (the crop being sown in October, and harvested about April), and that in the district with the earliest harvest, the earlier part of this period was more important than the later. My own observations are in general agreement with these, except for the somewhat natural proviso that it is the humidity of the air within the crop, rather than the normal air humidity, that is of consequence. In a field at Pusa, sown throughout with the same variety of wheat, rust was severe in an area with a dense stand, and almost absent at an end of the field where the growth was light. In the dense crop, the air humidity was nearly 20 per cent. higher than in the light crop as tested by running an experiment lasting between seven and eight hours on a dull day. The temperature averaged about 3 degrees C. lower in the dense crop. Soil moisture determinations showed that there was much more moisture under the dense crop, especially from the 3rd to the 6th foot, and this was really the cause of the good growth, as soil moisture is the chief limiting factor in wheat-growing at Pusa.

Lauritzin has found that the black stem rust of wheat is closely dependent on air humidity for its powers of producing infection. He only succeeded in uredospore inoculations at relative humidities approaching 95 per cent., and failed completely at 92. Dew-fall is ordinarily sufficient to provide the necessary moisture at night, but is light at Pusa in the spring, and is not enough to give much rust in sparse stands, except in years of high humidity and frequent ground fogs, at the season mentioned. Peltier has shown that when germination is below 10 per cent., very little infection can be caused by the uredospores of this rust.

In Italy, it has been noticed that either a drying-out of the soil or a sudden rise in temperature, makes wheat susceptible of mildew (*Erysiphe graminis*). Rivera believes that this is due to a lessening of turgescence, and is therefore a reaction of the host plant and not of the parasite. He states that it is particularly marked in rich soils, where the plants have a relatively restricted root development, and would naturally suffer from shortage of water or excessive transpiration. Against this is the fact that in India the cereal mildew is worse under shade. I believe that most experimenters with cereal cultures in houses have experience of the greater prevalence of mildew under such conditions than in the open. More diffuse light, and the absence of air currents, appear to be the causes, and exposure to light and ventilation I have found to be a remedy. But the action of the different factors does not seem to have been studied, though they are sufficiently marked to offer hope of exact evaluation.

To turn to a disease not caused by a fungus, but interesting as illustrating a sensitiveness to humidity of the same general order, the Indian rice disease, caused by the eelworm (*Tylenchus angustus*), may be mentioned. It occurs with great severity in an area at the head of the Bay of Bengal. Injury is confined to the parts of the plant above the water in which the crop grows, owing to anatomical structure, and to reach these parts the worm must leave the water and climb up the plant. It is a pure ectoparasite, never entering the tissues. Experiments proved that it is able to move on a dry surface, provided the relative air humidity is above some point between 90 and 95 per cent., probably near 93. Above this point, the worm has the power of condensing water from the air, and it moves in the film surrounding it by a snake-like method of progression. Neither feeding nor copulation are possible under totally immersed conditions, but only in damp air between 90 and 100 per cent. relative humidity. Below 90 the worm can neither move, nor feed, nor reproduce. Hence the disease is confined to areas of maximum air humidity during the summer and autumn months, the period when the rice is in a susceptible condition. It has been possible to correlate the meteorological data from six reporting stations within the affected area with the distribution of the

disease, and to forecast that the greater part of the rest of India, excluding part of the Irrawadi delta and some other smaller areas, is not liable to become permanently infected.

The smuts of sorghum, particularly the grain smut (*Sphacelotheca sorghi*), annually cause damage estimated at a million sterling in western India. They are also prevalent in the Punjab and submountain districts of the north-west, but are unknown in the eastern half of the Indo-Gangetic Plain. One of my students, Mr. G. S. Kulkarni, worked out the temperature relations of this disease. We found that sorghum has its optimum temperature for germination at the high figure of 37 degrees C. At this temperature (with suitable moisture), the seedlings were up in two days. At 20 degrees C., they took about twice as long; and at 16 degrees C., about three times. The spores of the smut fungus, however, have their germinative optimum about 20 degrees C., and germination falls off rapidly above 30 degrees, giving only 1 or 2 per cent at 37 degrees C. Infection is only possible during the earliest stage of seedling development. At high temperatures, not only is this stage shortened, but the smut spores germinate feebly. In incubation experiments, we failed to get any infection at 40 degrees C., whereas at 25 degrees there was 50 to 60 per cent. In western India, the main crop is sown in June-July, at a time when the temperature is from 21 to 27 degrees C., and naturally suffers severely. A second crop is often sown at a period when the temperature is over 30 degrees, and this gets relatively little smut. In a comparative experiment made by Mr. Kulkarni, seed grain was mixed with spores and divided into several lots. One of these was sown at Jacobabad, with an air temperature of 36 degrees to 40 degrees, and escaped infection. Another, sown at Poona, at 25 degrees, gave 65 per cent. smutted ears. At Pusa, in the eastern half of the Gangetic Plain, the temperature at sowing time ranges up to 38 degrees; smut is absent, and the artificially-infected grain remained free; but we have succeeded in getting a smutted crop by selecting abnormally cool periods for sowing infected seed.

Wheat bunt is a similar case. It is prevalent in Kashmir and along the Punjab foothills, occurs in the Punjab plains, but is unknown further east and south in many million acres of wheat. Bunt spores have a low optimum for germination. They germinate well between 7 degrees and 18 degrees C., and usually fail to germinate above 25 degrees. In central and eastern India, the temperature at sowing time is usually above 25 degrees (it is sometimes necessary to delay sowing to allow the soil to cool after the September heat, though wheat will germinate above 30 degrees), while in north-western India the temperature at sowing time is lower. In the latter case, the bunt spores on the grain can germinate and infect the seedlings, whereas

further east and south it is too hot. Hungerford, under Idaho conditions, found the maximum degree of infection was obtained with a temperature of 9 degrees to 12 degrees C., combined with fairly high soil moisture.

Maize smut (*Ustilago zeae*) has much the same distribution in India as bunt, but its temperature relations have not been worked out. It is almost certainly a similar case.

In comparing crown rust of oats with mildew, Fromme found that he could get no infection with the former below 80 per cent. relative humidity, and only 6 per cent. at 93, taking infection at saturation to be 100. With the mildew, on the other hand, he got good infection at from 75 to 80 per cent. relative humidity.

I think enough has been said to indicate the interest and importance of the study of the relation between meteorological conditions and cereal diseases.

Attempts are being made in various countries to accumulate information of this nature, with a view to practical application, though as yet no special attention seems to have been paid to the cereals.

In France, there has been for some time in operation a system of meteorological notification from a central station at Montpellier, fed by over 30 recording sub-stations, of the dates on which vine diseases are likely to appear, and thus when spraying or sulphuring may be necessary. The notifications are based on a knowledge of the conditions influencing germination of the spores and infection of the vine, incubation in the tissues, and subsequent external sporulation. It is possible to predict the latter seven days in advance, which gives ample time for protective measures.

In Italy, and parts of Germany, somewhat similar arrangements are made for notifying viticulturists when they should spray, and in the former country the service has been brought to a high state of efficiency.

In Germany, the Biologische Reichsanstalt, in Berlin, has established a laboratory for meteorology and phænology, and has commenced to collect data by means of a system of report cards issued throughout the country. A limited number of characteristic diseases are selected, and an attempt is being made to correlate their incidence and regional distribution with the annual fluctuations of the seasons.

The United States Weather Bureau is carrying on investigations to determine the effect of current meteorological factors on the growth of vegetation. As a part of this, systematic meteorological and phænological records are being kept at a number of centres, with a view to determining the critical periods in the growth of crops, and in the development of injurious insects and fungous diseases.

But I do not, myself, believe that the most rapid and assured progress will be made in this way. The evidence available points to the advisability of beginning at the other end—of first establishing the temperature and humidity requirements of the parasite and host singly and together—and only then, with the exact information thus gained, seeking the correlation with meteorological data.

The physiological apparatus required for these studies has now been brought to a considerable degree of perfection and simplicity of application in the United States. Those of us who have seen these studies in progress have learnt that, once the apparatus is installed, it is comparatively easy to run a test.

The co-operation of the meteorological authorities will be required the moment it is sought to translate the results of exact studies of the nature I have indicated into practical application. I do not know how far the data at present recorded in this country are sufficient. In India they are not, and neither the temperature nor, especially, the humidity records are suitable for this purpose. Even where the observations are taken three times daily a correction formula appears to be required. Several such formulæ have been worked out for the mean temperature, but their application differs in different localities. For the purpose here indicated, not only the mean but the duration of the daily extremes may be of the utmost importance, a low minimum, if sufficiently prolonged, being perhaps enough to start an attack, though it might not be revealed in the daily mean if counterbalanced by a hot day. Hence it may be necessary to take continuous records, and this is what we started doing in Pusa when we found our ordinary readings insufficient.

I have not aimed at covering all the aspects of this subject. Many points of great interest have been omitted as, for instance, the fascinating question of the influence of climate on the over-wintering of the rusts. There must be several here to-day who can add other aspects and new points of view based on their own experience. Mine has led me to be sceptical of the value of field observation, unless controlled and elucidated by the more exact methods of laboratory research.

34. Methods of Reducing Losses from Black Stem Rust of Wheat.

By Professor E. C. Stakman, Plant Pathologist, University of Minnesota, and Pathologist in Cereal Investigations, United States Department of Agriculture.

There are three general methods for reducing losses caused by cereal rusts: (1) by destroying the source of inoculum; (2) by making conditions unfavorable for the development of rust; and (3) by developing resistant varieties. Practically this includes the eradication of alternate and wild grass hosts, soil management, and plant breeding.

In regions in which the alternate host of a rust fungus is prevalent, and rusts heavily, it should be eradicated. One need only observe the devastating effect of the common barberry in Europe and northern United States to marvel at the stupidity of man in permitting the bushes to be distributed, and to remain in grain-growing regions. There is excellent evidence that the severity of stem rust attacks increased as barberry bushes were distributed in Europe. In fact, there can be no question but that the stem rust cannot be destructive in many regions of Europe unless there are many barberry bushes. England, Denmark, Holland, parts of Germany, France, and certain of the central European countries, have eradicated barberry bushes more or less completely. And black stem rust ceased to be destructive. England, Denmark, and other countries certainly solved the stem rust problem by eradicating the common barberry. This is not propaganda, but a scientifically accurate statement.

An extensive barberry eradication campaign now is being carried on in the United States. The early colonists brought with them seeds or plants, and planted them near their new homes. The bushes escaped from cultivation, until now the common barberry is thoroughly established as a wild bush in the New England States, in many of the middle Atlantic States, and even in some of those of the Upper Mississippi Valley. There is perfectly clear and conclusive evidence that these bushes are the principal source of inoculum in much of the soft red winter wheat region, and in the hard red spring wheat-producing states. Thirteen states of these two regions now have legal provision for the eradication of the bushes. The Federal Congress has appropriated as much as $350,000 a year for the eradication work, State legislatures have made appropriations, and milling interests and other commercial organizations have been contributing about $50,000 a year to assist in the work. The task is appalling, but it must be finished. How much simpler it would have been if the barberry never had become established. The common barberry should not be tolerated in grain-growing regions.

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37. The Helminthosporium Foot-rot Disease of Wheat in New South Wales.

By H. J. Hynes.

[*Abstract.*]

Mr. Hynes pointed out that the late Mr. C. O. Hamblin was the first in Australia to show that Foot-rot was due to a specific fungus. This serious disease was found to be present on over 150 different wheat varieties in addition to oats, barley, rye, and a few grasses. Mr. Hynes outlined the extensive investigations now being carried out on this disease at the University. Already he has succeeded in isolating five different strains of the fungus which causes the Foot-rot disease in cereals.

38. *Recent Investigations of the Flag Smut Problem, by R. J. Noble, D.Sc., New South Wales Department of Agriculture.*

DISEASES OF MISCELLANEOUS CROPS.

39. Virus Diseases of Plants.

By Dr. E. J. Butler, Imperial Bureau of Mycology, London.

The convenient term "virus," borrowed from the animal pathologists, is used in connexion with plant diseases to denote a causative agent of disease of whose nature we have no exact knowledge by ocular means, but regarding which we have evidence in many cases that it is so small, in some of its stages, as to belong to the so-called ultra-microscopic organisms, and to be capable of passage through the ordinary bacterial filters, such as those of Chamberland, Berkefeld, and the like.

It is, perhaps, not very generally known, at least by animal pathologists, that the first demonstration of the existence of a disease of this type was made by a plant pathologist, Ivanovski, in the case of the mosaic disease of tobacco, in 1892. Six years later, about the time that the first case of an animal disease of a similar nature was recorded by Loeffler and Frosch (foot and mouth disease of cattle), Beijerinck fully confirmed Ivanovski's work. Since then, over 100 species of plants, belonging to over 30 different families, have been found to suffer from diseases of the mosaic type. At the same time, many human and

animal diseases of great importance, including such a terrible scourge as typhus fever, have been shown to be caused by viruses of one kind or another.

The symptoms in all these mosaic diseases are more or less alike. The most striking, to which the name is due, is a mottled appearance of the leaves, caused by the development of mingled light and dark-green patches or streaks on them. Another frequent symptom is a crinkling or blistering of the leaves, due to uneven growth of different areas or surfaces of one and the same leaf. Both these characters are well shown in the specimens exhibited, the sugar-cane and nasturtium mosaics having the mottled appearance only, while the bean leaves are crumpled and blistered as well. Other symptoms are also found in certain of these diseases, the chief being such as result from deep-seated disturbances of normal nutrition and growth, stunting and malformation of stems, leaves and fruit, and changes in growth habit. Although the virus has not actually been filtered in a number of mosaic diseases, there is considerable evidence that they are all systematic diseases, caused by something present in the sap, and this has been shown, in several cases, to survive passage of the sap through bacterial filters. In the uniformity of their symptoms, methods of transmission, and other characters, there is ample reason to warrant the belief, now generally held, that, etiologically, the mosaic diseases of plants form a homogenous group.

Few definite necrotic lesions have been, so far, found in plants affected with mosaic disease. The tissue changes observed are mostly only those due to hypertrophy, atrophy, or imperfect differentiation. In certain cases, however, necrosis of groups of parenchyma cells has been observed, and has been associated with foreign bodies in these cells by at least one worker, as will be seen later on.

Another, much smaller, group of virus diseases consists of those of which the leaf roll of potato is the best known type. In this group there is no mottling of the leaves, but the latter show a peculiar longitudinal rolling of the edges, accompanied sometimes by a crinkled or drawn-thread appearance along the veins, and a dry, harsh texture. The latter symptom is associated with a gorging of the leaf cells with starch, the starch formed during the day not being removed during the following night. As a result, the growing parts receive insufficient nourishment, and tend to bear small leaves, and have short stem internodes. In several of these diseases a well-defined necrosis of the phlœm is found, in others, such as peach yellows, this has not yet been observed, though leaf roll is a constant feature of the early stages of the disease, and is its most reliable symptom. In the spike disease of sandalwood in India, phlœm necrosis has not been reported, and there is little rolling of the leaves, but in other respects the similarity to peach yellows is considerable.

Few of the virus diseases of plants are rapidly fatal. The most common effect is a debilitating one, marked in the case of potato leaf roll and mosaic by a reduction in the crop, which is progressively less each generation. Hence the general term, "degeneration diseases," is sometimes applied to them, and many of the supposed cases of degeneration or "running out" of crops under long-continued cultivation are now known to be due to these diseases. The relative scarcity of necrotic lesions, and the delayed injury to the host plant are amongst the reasons for believing that the causative agents in these diseases belong to that highly adapted class of parasites that enters into symbiotic relationships with the host plant, and avoids, as long as possible, causing such injury as would reduce the supply of living nutrition. Amongst the higher plant parasites, the rusts and smuts are the best known instances of this. Premature death of the host causes death to the parasite, since ability to live as a saprophyte is almost or entirely lost. No rust has been artificially cultivated apart from its host, and, except for their size, the same arguments against their existence as separate organisms could be advanced as have been used in the case of the cause of the virus diseases. Merely from the general characters of this latter group, there is reason to suspect that their causal organisms may be as incapable of development apart from the living host as the rusts are, and to anticipate that to cultivate them in artificial media would be beyond our present resources.

Much is known regarding the transmission of these diseases. In every case that has hitherto been investigated, they can be communicated to a healthy susceptible plant by grafting or budding from a diseased plant, provided organic union is secured. In certain cases, such as peach yellows, this is the only method of transmission that has been proven, though there is ample evidence of some other method in nature, and, indeed, epidemic spread sometimes occurs. In spike disease of sandalwood, artificial transmission by budding, and natural transmission through the haustorial connexions (sandalwood being a root parasite) have been proven, but in this case also spread in some other manner is strongly suspected, and it may be that it is carried by insects. In no case that has been found up to the present is mere contact of unwounded surfaces sufficient to permit of infection. The plant cuticle seems to offer an impassable barrier to the virus.

The incubation period in graft inoculations is sometimes very long. In peach yellows, when the inoculation is made by taking a bud from a distant part of a tree, showing only a single infected twig (all parts of the tree are known to contain the virus in such a case), incubation periods up to five years have been recorded.

The next most general method of transmission is by insects. This method is fully established in many diseases of the mosaic type, and in some of those characterized by phlœm necrosis, and it is probably general in both these groups.

In the "curly top" disease of beets, only one insect (*Entettix tenella*) has been found which can transmit the disease. Furthermore, the insect is only able to infect a healthy plant after a certain period (varying from 4 to 48 hours) has elapsed since it has fed on a diseased plant. This would seem to be clear proof that, in this case, the insect is a necessary secondary host, like the mosquito in malaria.

In spinach blight the causative agent is carried by aphids (as in a great many mosaic and leaf roll diseases), and is transmitted to the progeny of aphids that have fed once on a diseased plant, down to at least the fourth generation.

The third method of transmission known is by inoculating with the juice of infected plants. This is only certainly known to occur in the mosaic group, but has been proved in over a dozen of these. The failure in other cases, especially in some of the leaf-roll type, may be due to the relatively coarse method of inoculation adopted as compared with that used by aphids. The latter cause a minimum of injury to the plant cells by their fine probosci, whereas a needle prick kills the cells which are entered. Possibly, as in the rusts, injured cells are unable to provide the parasite with the conditions necessary for its growth.

In certain mosaic diseases, however, especially those of tobacco and cucumber, juice inoculations succeed readily. In the former case the virus is found in every part of the plant except the embryo of the seed. Its absence from the latter is correlated with the failure that most observers have reported to show that the disease is seed-borne. In the cucurbit and bean mosaics, however, seed transmission is known.

Most of these cases are true systematic diseases, but there are some exceptions. In sugar-cane mosaic, for instance, the virus is often confined, for a considerable period, to the tops of infected canes, and perfectly healthy shoots may be taken from the base of the stool.

Immune species are known in several genera that suffer from mosaic, e.g., *Nicotiana*. Inoculation of an immune species may set up a destructive tissue rot, though no other symptoms appear, and rotting is not a symptom of mosaic on a susceptible plant. Here again we have a suggested explanation in the possibility that the parasite can only flourish in living tissues, and may destroy itself in destroying the cells on which it feeds. Immunity may really be due to hypersensitiveness of the host, as is known to be true of certain varieties immune to the rusts. In other cases "carrier" species are known, that is certain varieties of host may show no symptoms on being inoculated, but their juice is infective, and will cause characteristic symptoms to appear if inoculated back into a susceptible host. Climatic conditions may sometimes even cause an infected susceptible plant to develop normally and show no symptoms; infected potatoes give apparently healthy plants and bear a good crop in certain parts of Canada, but the symptoms reappear when the plants are moved to another climate.

In considering the nature of the virus, the tobacco mosaic is, perhaps, that of which we have most knowledge. In this case the virus can be filtered through the Chamberland, Berkefeld, and Kitasato filters, but the fine-pored kinds pass it less readily than those with coarser pores, as is shown by the relative virulence of the juice after filtration. It will not diffuse through parchment or celloidin membranes, nor through moderate thicknesses of stiff agar. Working with a series of filters differing in porosity, it has been possible to find two members of the series, one of which allowed the virus to pass at full strength, while the other gave a very weak filtrate. Assuming that the size of the particles is near the limits which the latter filter allowed to pass, and using this filter on colloidal organic solutions, an attempt has been made to estimate the size of the virus particles. They were found to behave approximately as do colloidal particles of fresh hæmoglobin prepared by standard methods. The latter are said to be about 0.03μ. in diameter. We can see particles of, perhaps, one-tenth or one-fifth of a micron, but one-thirty-third of a micron is far below the limits of the best microscopic vision.

Other characters of the tobacco mosaic virus of interest in connexion with its nature are its behaviour to antiseptics and its keeping properties. The details of the elaborate work done on these points would take too long to enter on here. All that need be said is that though the tobacco mosaic virus can be kept with unimpaired virulence for long periods in the dried leaves or even in the juice allowed to putrify, there is ample evidence in several cases that there is at least no saprophytic life in the soil. Sugar-cane or potatoes may be immediately planted in the holes from which diseased plants have been removed without any risk of infection.

To sum up the evidence that these diseases are caused by living organisms, we have the following considerations:—(1) The continued extension of some of them to new areas, as in the sugar-cane mosaic and spike disease of sandalwood. (2) The rapid multiplication of the virus in the plant, and its passage to all, or nearly all, parts. Full systematic infection may occur in herbaceous plants in a relatively short time, and a tobacco leaf with its stalk may be traversed in three days. (3) The capacity of the virus to cause systematic infection in very minute quantities, and to stand very high dilution. The quantity of juice contained in a few leaf hairs snipped off with fine scissors is sufficient to cause infection with tobacco or cucurbit mosaic, while dilutions of 1 in 10,000 may still be infective in a certain number of cases. (4) The power of unlimited transmission to new healthy individuals. (5) The transmission of these diseases by insects, and other points of similarity to various diseases of man and animals now generally admitted to be caused by minute, and often ultramicroscopic

organisms. (6) Above all, the discovery in the curly top disease of beets that the insect is not immediately infective after feeding on a diseased plant.

Various observers have noted the presence of foreign bodies, which have been taken for living organisms, in certain of these diseases. As long ago as 1903, Ivanovski reported the presence of small granules, 0.3μ. in diameter, in the cells of tobacco leaves suffering from mosaic, and regarded them as minute bacteria. He also described other, larger bodies, which he thought were products of cell degeneration.

In 1921 Kunkel found large bodies, which he likened to the *Neuroryctes* of rabies, or the *Cytoryctes* of variola in maize affected by mosaic in Hawaii. Subsequently he found similar bodies in *Hippeastrum*, sugar-cane and Chinese cabbage mosaics. They were found in necrotic parenchymatous tissues, and in sugar-cane are stated to be confined to a region near the growing point, and disappear as soon as the cells containing them break down.

In 1922, Palm, in Java, and Dickson, in Canada, independently described granular bodies in the cells of mosaic areas in tobacco. Both agree in likening these bodies to the so-called chlamydozoa that have been described in certain virus diseases of man, rather than to bacteria, as Ivanovski had done. Large amœbiform bodies were also seen by Palm, and other bodies of various types by Dickson, but were considered to be products of cell reaction. In England, K. Smith reported a little later the presence of large amoebiform bodies in the tissues of potato mosaic. These had a certain resemblance to those described by Kunkel.

Lastly, Ray Nelson, working in Michigan, has recently described the regular occurrence in the phlœm tissues of tomato, bean and clover mosaic, and in leaf roll of potato, of quite large organisms of a protozoal type, sometimes up to 35μ. in length. These have not been accepted as genuine protozoa by certain workers in the United States and England, who have endeavoured to duplicate Nelson's results, and the matter is still in dispute. However that may be, it is obvious that, if the estimates of the size of the infective particles given above is even approximately correct, none of these bodies can be regarded as the filter-passing stage of the causative organisms. We must suppose, if we are to admit them to be the cause of these diseases, that they give off much smaller propagative particles, such as have already been stated to be given off by certain bacteria. The writer has advanced the view that the supposed causative agents of some of these virus diseases of plants, as described and figured by certain of the above observers, show a great similarity to the bodies described under the name of *Rickettsia* in typhus and Rocky Mountain fever, and in the insects by which these diseases are transmitted.

Workers on human and animal pathology have advanced our knowledge of the causes of this great class of diseases beyond the stage reached by plant pathologists. The latter may, however, claim to have been amongst the first to convict insects of playing a primary part in natural infection, and have most fully studied this means of transmission; they have proved that these diseases may be hereditary in both the plant host and the insect vector; they have shown that the unwounded plant surface does not permit of infection; that a saprophytic life of the organisms is highly improbable; and that they are often so closely associated in some way with the living tissues of the host that infection appears only to be possible when organic union between diseased and healthy tissues is secured. These must be regarded as very important contributions to comparative pathology.

40. Some Aspects of the Bunchy Top Disease of Bananas.

By Dr. G. P. Darnell-Smith, Biologist, N. S. W. Department of Agriculture.

The "Bunchy Top" disease of bananas made its appearance in the Tweed River district a few years ago. It has now spread throughout the district, and a large number of plantations have ceased to be profitable owing to this disease. The disease may make its appearance with comparative suddenness in a plant not six months old, or in one two years old or more. The main characteristics of the disease are that the plant ceases to grow; the leaves, instead of expanding normally, assume a more or less upright position, and become bunched, hence the name "Bunchy Top," or "Cabbage Top"; the apex of the pseudo-stem appears more or less constricted, and a plant that is sufficiently mature to produce a bunch, will often be found with its bunch more or less jammed in the constriction; the fruit is short, brittle and sweet, the tissues of the leaves and petioles are brittle; at the base of the leaf sheaths which are normally of an even colour, dark green lines are visible; finer green lines are visible in the midrib and in the lamina of the leaf. If the plant has pushed forth an inflorescence, the apices of the bracts subtending the male flowers will be green instead of red. Section of a healthy corm shows a creamy surface rapidly darkening to bluish black from reaction of tannin; section of a "Bunchy Top" corm shows fine streaks, yellow, red, brown, or black, and the tannin reaction is not so well marked. The sap from a healthy corm is at first of a pale yellow colour; the sap from a "Bunchy Top" corm is of a distinctly reddish tinge when first collected. The roots of a healthy corm are white or light purple with numerous laterals and abundant root hairs. The roots of a corm that has been diseased for a short time are always dead

at the ends, and the laterals are dead also. The dead ends are at first black, and the blackening proceeds steadily to within an inch or two of the corm; the central cylinder is brown. Sometimes the blackening makes its appearance at two different points on the root, the sap between these two points then becomes alkaline, whereas the sap of healthy and of diseased plants is normally acid. The interior of a "Bunchy Top" plant is often a putrid mass. The apex of a healthy leaf is, when young, covered with a small cap, which falls off as the leaf expands; in a diseased leaf the cap falls off but the leaf does not expand, and rain, collecting in the leaf funnel, forms a stagnant pool in the interior, in which decay is soon set up. Owing to the constriction of the pseudo-stem at its apex the internal young tissues are frequently forced together, and become wrinkled and folded. A condition which is to be distinguished from "Bunchy Top" is sometimes brought about by drought. The pseudo-stem becomes constricted at its apex, the plant cannot force out the bunches of bananas, but growth continues from below, and as a consequence, the true stem becomes twisted, or, in popular parlance—it gets "goose-neck." Another condition which is to be distinguished from "Bunchy Top," is the attack of a corm by some fungus when the corm has been planted immediately over some old tree root. In neither of these cases is the infallible sign of "Bunchy Top" present, viz., the green streaks previously referred to in the midrib of the leaves. The presence of a faint green streak in the midrib of the leaf is the first and infallible sign that the plant is going to develop "Bunchy Top."

In the Tweed District, three classes of soil may be distinguished. Scrub land, that is, land upon which softwood trees have been growing. This is regarded as the best land for bananas. Forest land, upon which hardwood trees have been growing. This is regarded as the least suitable for banana growing. Bastard scrub is intermediate between these two. Scrub land gives the heaviest yield of bananas, but, when "Bunchy Top" disease makes its appearance, the disease seems to spread more rapidly than in the other cases. The soil is a well-leached clay, with a very compact subsoil. Corms are generally planted in virgin land, immediately after the scrub has been felled and burnt. The disease frequently first makes its appearance in the neighbourhood of old tree stumps, and spreads from such a centre, but often a number of apparently healthy plants are found around a diseased one. It does not appear to occur to such a marked degree upon land that has been for some years under grass after sugar-cane, and that has previously been well worked. Plantations kept absolutely free from weeds have developed the disease as well as those in which the cultivation has not been so thorough. As nearly all the plantations are on steep hillsides, cultivation is, for the most part, carried out with the hoe. If a plant which has developed "Bunchy Top" is dug out and a fresh one planted in the same place it eventually develops "Bunchy Top". Superfluous

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responsible, and, secondly, that it is his subject which ultimately suffers. It is sometimes argued that it is not the business of the scientific man to deal with matters which are in the main administrative, and in theory there is much to be said for this doctrine, which is certainly convenient for the biologist himself. In practice, however, there is one overwhelming reason why the scientific man must not fall in with this attractive point of view; it is that he alone has the specialized knowledge which will enable him to detect incompetence and signs of dishonesty, the latter being patent to him though carefully hidden from the world at large. It is quite unreasonable to expect the lay administrator or clerk to decide upon questions relating to the presence of pests or the health of plants, and equally so to judge of the competency of an inspecting official on a technical point. Yet only too frequently the responsible biologist lays down a policy, and leaves it entirely to others to carry out, overlooking the fact that they are not competent either in technical knowledge, or perhaps numerically, to undertake the task.

Two suggestions are therefore made. First, that responsible biologists should frame their policies so as to be within the scope of the machinery available, remembering that success will depend on a sufficiency of technically trained men, and that inadequate wages are the greatest incentive to dishonesty. Secondly, that they should never allow themselves to lose touch with the practical details and difficulties of the work, which are quite as important as the framing of the original policy.

The foregoing suggestions are made with some hesitation owing, as has been stated, to great variations in conditions found in different counties. Nevertheless it is hoped that they will serve to indicate that without any sacrifice of principles something can be done to ameliorate trade interference, and so allow of the peaceful evolution of phytopathological legislation.

51. Some Observations on Plant Quarantine.

By Dr. E. J. Butler, Director, Imperial Bureau of Mycology, London.

I propose to offer some brief and rather disconnected observations on certain aspects of the attempt to limit the spread of plant diseases from one country to another. These observations are made rather with a view to provoke discussion than as a serious contribution to the subject.

For many years it has seemed to me that, in order to restrict the spread of diseases to new areas, it is essential that we should keep clearly before us the means of dissemination possessed by parasitic fungi and bacteria. Fungi, for instance, can be disseminated either by means of their mycelium or their spores.

Ordinarily we are inclined to regard mycelial dissemination as rather a minor or secondary method, and this is, no doubt, true as regards the dissemination of most fungi over short distances, and from plant to plant, as when epidemic outbreaks of crop diseases occur in any particular area. But mycelial dissemination assumes quite another aspect when we have to consider the conveyance of parasites over long distances, as across the oceans. In this case, we find that the power possessed by the majority of fungi of living in a vegetative condition as mycelium in the tissues, that is, immersed in the interior of the host plant, is one of very great and serious concern to those responsible for keeping out foreign plant diseases.

Such mycelial growths in the interior tissues are out of reach of any treatment by fungicides or the like, and are only rarely capable of being killed by the application of heat without, at the same time, killing the host plant. Hence it is that the mycologist has not been able to devise any satisfactory method of treating consignments of plants on arrival at a port of entry so as to free them from fungus diseases.

A large number of the most serious diseases of whose spread we have authentic records have undoubtedly been carried in this manner. As instances, one may mention the potato blight, the white pine blister rust (*Cronartium ribicola*), and many sugar-cane diseases (red rot, wilt, pine-apple disease, mosaic, Fiji disease, &c., &c.)

Of the same general type is the conveyance of parasites by means of resting spores in the tissues. This is the case in such instances as the wart disease of potatoes (*Synchytrium endobioticum*); finger and toe of turnips (*Plasmodiophora brassicæ*), downy mildews, such as the cereal and sugar-cane *Sclerosporas*, and many *Ascomycetes*, which form spores in closed cases (*perithecia*), which are immersed or partly immersed in the tissues, and resist the application of fungicides.

Now, the trouble about this form of dissemination is that it is very frequently impossible to detect either at the time of packing or on arrival. The consignment may appear healthy while containing the parasite. For instance, the white pine blister rust has an incubation period of many months, and the swellings may not be visible until long after infection has succeeded. So also sugar-cane wilt was found in setts imported to Pusa, India, under every possible precaution from the Department of Agriculture, in the United States of America, and probably gained access in the form of spores at the moment of packing, these spores subsequently infecting the setts during transit.

Still less possible is it to detect in a consignment, we will say of grain, the presence of loose spores. In the majority of cases the loose spores produced by fungi serve the object of immediate, short-distance dissemination, and do not live long. But we have many cases, such as the teleutospores, and even sometimes the uredospores of rusts, and the spores of smuts, in which these reproductive bodies live long enough to survive passage round the world. In most such cases, however, it is possible to free the consignment from casual adhering spores by disinfection. Where the consignment is a bulk one intended for consumption or manufacture, disinfection is obviously impracticable, and is ordinarily not required, as the chances of the parasite escaping and infecting the crops of the importing country are slight. But where the consignment is intended for growing, it cannot be too often emphasized that it is madness not to disinfect before sowing. Most seeds will stand disinfection in 0.25 per cent. formaldehyde for two to four hours, and repeated experiments have shown that this is usually effective against parasitic fungi.

This is, then, the limitation, for practical purposes, of our means of destroying parasitic fungi after they have reached the importing country, and before they are released to the consignee. They are, as you will see, slight. They do not guard against parasites capable of being conveyed in the interior of the hosts (grafts, cuttings, &c.), and are only effective against certain classes of parasites likely to be carried chiefly as loose spores on the surface of seeds and the like intended for planting.

Hence it is that mycologists have been forced to give considerable attention to the possibilities of securing that none but healthy consignments of plants or plant parts, other than those intended for consumption or manufacture, are received in the ports of importing countries, or alternatively, that all plants or plant parts intended for growing should be planted in a quarantine ground, and grown under supervision until it is clear that they are free from diseases not present in the importing country.

Most countries now maintain an inspecting staff of qualified persons, and issue certificates vouching for the freedom of exported consignments from specified diseases. The strides made in this direction during the past ten years are by no means fully realized in many countries. Errors still occur, but the number of cases in which diseased consignments are sent out from such countries as Holland and England is small, and is steadily diminishing. Naturally, I am not referring to common diseases of world-wide distribution, such as crown gall or apple scab ("black spot": *Venturia*), regarding which certificates are not usually required.

But there still remain cases where it is impossible on the most rigid inspection to detect the occurrence of spores or buried mycelium in a consignment. Hence we must admit that no system of inspection and certification can provide a complete guarantee against the introduction of disease.

What are we to do? Are we to accept the risk in view of the advantages to be gained by the introduction of new or improved varieties? Or are we to prohibit the importation of whole classes of plants which contain this element of risk, and aim at becoming completely self-supporting in the matter of horticultural and crop improvement, as is the trend of recent legislation in the United States of America? Or, finally, are we to compromise by taking the risk when it is worth while, and then endeavouring to safeguard ourselves by growing the imported produce under strict supervision in quarantine until danger has disappeared?

These are the questions which it seems to me we must face. What guiding principles can be discovered with a view to helping us to make up our minds? This is a matter for much fuller discussion than it appears to have as yet received. I will mention a few of the principles which appear to me to require consideration, and I have no doubt that others will occur to many of us.

1. When a country is endeavouring to establish a new crop, or to develop one beyond the experimental stage by a full testing of varieties in order to find those most suitable (as in the case of the present development of cotton growing in Australia), the most rigid precautions are required. No imports should be allowed with which there is a risk of introducing a major disease or pest unless there is absolute certainty that effective disinfection or quarantine can be secured. No precautions of this nature can be regarded as hampering trade, as no trade exists in such cases, as a rule.

2. When a staple crop of a country is threatened by the introduction of a disease not already present in that country, rigid precautions are again justified. But here the introduction of a new variety may be of such importance as to counter-balance the risk, and lead to introduction

under such precautions as may seem to eliminate it. The experience of Hawaii in regard to sugar-cane importations deserves study in this connexion.

3. When a crop or horticultural plant is not of major importance in a country, trade considerations may be allowed greater weight. Each case requires to be taken on its merits, and to bar whole classes of horticultural produce is often unwise. Our object should be in all such cases to hamper the interchange of horticultural produce as little as possible.

In many of these cases it seems that much more might be done by means of suitably located quarantine grounds, under adequate supervision, than has hitherto been attempted. I think the time will come when properly equipped quarantine grounds for this purpose will be an essential part of the machinery of the plant protection service in all countries. They have the advantage over partial prohibition, that is prohibition of imports entirely from certain localities, or in regard to certain crops, that they will permit of some information being gained as to the possibilities of particular diseases becoming established in a destructive capacity in a new area. Fire blight, for instance, has probably been introduced into England, but has failed to become established, probably owing to climatic limitations. Isolated quarantine areas would at least give some indication of the virulence of newly imported diseases.

Another matter which requires consideration, because it is directly helpful to the officials of any country charged with the duty of protecting that country from introduced disease, is the need for an accurate knowledge of the distribution of the major and minor diseases of plants in other countries. I include minor diseases on account of the well-known fact that what is a minor disease in one country may become a major disease in another. It is obvious that it would be of the greatest value to your cotton officers to be able to turn up the records of any country from which they may wish to import cotton seed, and find at a glance what cotton diseases occur in that country. That there has not been a greater clamour for the undertaking of plant disease surveys is merely another indication of the somewhat haphazard manner in which this whole question has been dealt with, and the very recent realization of its importance to the agriculture of a country. In the matter of plant disease surveys the United States of America has led the way, and the records are fairly complete for many of the chief crops in that country. England, Holland, Germany, and other European States are following with varying degrees of efficiency.

In conclusion, I would like to emphasize the need for a full, free, and frank discussion of this subject. Mycologists are naturally prone, in view of the great difficulties in detecting and destroying infected consignments mentioned above, to be tempted to become whole-hearted

prohibitionists. It is far easier to feel that you are doing your duty in safeguarding the crops of your country from the ravages of disease by absolutely prohibiting dangerous imports than by any less rigid procedure. But I think this attitude of mind requires very careful checking, so that each individual case may be examined on its merits to see how far the advantage in obtaining new or improved varieties counterbalances the risk, or how far trade interests require to be considered. Then, when a case for permitting imports appears to exist prima facie, the mycologist must give serious consideration to the limitations that should be imposed. These will generally involve a choice of the locality, and sometimes of the individual or institution from which the produce may be obtained, a fixing of the certificate or other guarantee of freedom from disease which must accompany the consignment, the inspection and disinfection of the consignment on arrival, and its removal to a quarantine ground for growth under supervision. It is in these directions, I believe, that a solution of this very thorny and difficult problem will ultimately be found.

52. The Importance of Horticultural Quarantine.

By Dr. Edward M. Ehrhorn, Chief of the Division of Plant Inspection of the Hawaiian Islands.

The traffic in plants and plant products, such as fruit, vegetables, seeds, and cereals has been going on for centuries. Many of our cosmopolitan pests have been distributed by the early navigators. The early whaling fleets carried many household pests from country to country, and these in turn have spread and become common property the world over. As the agricultural industries developed, such pests as attacked our field crops became very numerous owing to the increased area producing food for these pests, and enormous losses resulted. Worst of all were the losses caused by pests of foreign origin, as these were introduced without their natural checks, the parasites and predatory enemies. The United States of America Department of Agriculture several years ago issued a careful analysis of the losses caused by insect attack to the principal crops of the United States of America, showing that these losses amounted to upward of a $1,000,000,000 a year, a sum more than sufficient to meet the entire cost of the Federal Government expenditures. Taking the value of the crops of to-day, with the ever-increasing labour cost, this sum could, without doubt, be doubled. These estimates, as stated above, only touch on the ravages of insect pests, when, for a fact, losses due to such plant diseases as rust and smuts, mildews, blights, and many other diseases of plant life, would add considerably to the total. The losses in the United States are caused by a host of pests, insect and

1928

NYASALAND PROTECTORATE

REPORT ON

Some Diseases of Tea and Tobacco in Nyasaland

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Fig. 1.—View from Suruku Bungalow, Lauderdale.

Fig. 2.—Chitakali.

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INTRODUCTION.

The following notes are the outcome of a visit to Nyasaland between the 15th February and 31st March, 1927. This visit was undertaken at the request of the Nyasaland Government, mainly as a result of representations made on behalf of the tea planters in the Protectorate. It was natural, therefore, that most attention was given to the difficulties experienced in several estates in securing a full crop owing to the injuries caused by diseases of the tea bush, and the greater part of these notes refer to this matter. As much work as possible was done on these diseases on the spot and material was brought back to England for further study. A little time was available for observations on the chief diseases of the tobacco crop that were to be found during the period of my visit. These, however, were ascertained to be essentially similar to those occurring in Rhodesia and South Africa, where they have been the subject of considerable study, and there does not appear to be the same need for local investigation of tobacco diseases, in spite of the importance of the crop, that there is in the case of tea, in regard to which practically no work has been done in southern or eastern Africa. One tobacco disease, however, was found which had not previously been recorded in this area, and a somewhat detailed account of this is given.

The visit was timed to coincide with the rainy season, when it was hoped that disease processes would be showing their greatest activity. In general this hope was probably fulfilled, but I was unfortunate in not finding a severe attack of the tea canker attributed to *Macrophoma theæ*, either because some seasonal factors had diminished its activities or the measures undertaken the previous year on the advice of the Nyasaland Agricultural Department had effectively checked its spread. As this appears to be one of the diseases peculiar to Nyasaland the scanty opportunities for its study were regrettable.

The possibilities of tea cultivation in various parts of east and central Africa are at present attracting a good deal of attention. In the older tea countries of the East, extension is limited by scarcity of new suitable districts, insufficient labour, and the competition of other plantation crops. In Africa good land is abundant and other difficulties are not such as cannot be overcome. The Nyasaland tea industry is the only one of appreciable importance as yet, and its fate will be watched with interest by neighbouring colonies. Coffee was wiped out in the Protectorate by the depredations of insect pests. The cotton area is declining, and unless some method of controlling boll worms is found, they will prevent the extensive development of this important crop in a region that is otherwise very well suited for its growth. In the same way, the small but promising tea industry might well become unprofitable owing to the unusual prevalence of certain types of disease, but all of these, save one, are, I believe, susceptible to fairly ready control—and the one exception is, perhaps, only formidable because its origin is as yet unknown.

As there has been some little controversy about the earliest introduction of the tea bush into cultivation in Nyasaland, the following details may be of interest. The earliest attempt to introduce coffee and tea that I have been able to trace was in 1878, when plants of both species were brought out from the Royal Botanic Garden, Edinburgh (the Regius Keeper of which at that time was John Hutton Balfour) by Mr. Jonathan Duncan, a gardener of the Blantyre Mission of the Church of Scotland. All these died except one coffee plant, from which 400 seedlings were raised and these yielded 14½ cwt. of coffee in 1883. By 1891 the coffee industry was firmly established in the Mlanje district, and in 1901 the area under the crop was nearly 17,000 acres, but by 1925 it had fallen to 857, mainly as a result of an unsuccessful fight against insect pests. In 1888 a further attempt was made to introduce tea, Dr. Elmslie of the Livingstonia Mission of the Free Church of Scotland taking out plants in a Wardian case from the Edinburgh Botanic Garden, to the Keepership of which Isaac Bayley Balfour had just succeeded. These plants were handed over to Mr. Duncan at the Blantyre Mission Garden, where two survived and one is still to be seen. Seed from these early plants was taken to Lauderdale and Thornwood Coffee Estates in Mlanje, in one of which it was being planted in 1891, apparently by Mr. Henry Brown. In 1895 Mr. Alexander Whyte reported at the request of Commissioner (afterwards Sir Harry) Johnston and of the Royal Botanic Gardens, Kew, that "Tea of good varieties, Assam and China, has been introduced, but it is not yet cultivated to any extent. It grows luxuriantly and ought to do well

in some districts. . . . . I see no reason why it should not become a profitable cultivation." It is certain that Mr. Brown was responsible for the extensive early planting at Thornwood Estate, samples of manufactured tea from which, received for report in 1898, are preserved in the Museum at Kew Gardens. In 1904 there were over 200 acres under tea at Thornwood and 50 at Lauderdale, and in 1908 about 600 acres were planted and tea first figured in the Nyasaland exports. Subsequent introductions were in part, unfortunately, from Natal, whence varieties of low quality appear to have been derived. It is only of comparatively recent years that an attempt has been made to improve quality by the introduction of high class Indian seed, and most estates still show an excessively mixed population of bushes.

In the identification of Nyasaland tea diseases valuable pioneering work has already been done by Captain C. Smee, the Entomologist to the Department of Agriculture, and he and Mr. E. W. Davy, the Assistant Director, have had considerable success in getting an effective control of certain diseases, such as canker. Capt. Smee gave me invaluable assistance during my visit, the arrangements for which were made by Mr. E. J. Wortley, Director of Agriculture, without whom I would have been helpless. To these and the other members of the Agricultural Department in Nyasaland, my warmest thanks are due. I also received abundant hospitality and aid from members of the planting community, particularly from Mr. A. E. Shinn of the Ruo Estates, Ltd., and the Hon. W. Tait-Bowie and Mr. W. Morris Scott of the Blantyre and East Africa Co., Ltd., which I desire gratefully to acknowledge.

1. Diseases of Tea in Nyasaland.

The main tea district of Nyasaland extends from south-east to south-west along the foot of Mlanje mountain, for a distance of about twenty miles, at an elevation averaging 2,500 ft. above sea level. It rises from the Shiré Highlands, a wide and extensive plateau, on the south-eastern corner of which Mlanje is situated. The rainfall varies from about 70 to 90 inches, diminishing as the western extremity of the district is approached and also as the ground falls away from the mountain towards the plain: in general it is greater the nearer the mountain face is approached. In the western half of its extent the main mass of the mountain rises abruptly from a moderate to steep slope at its base. Towards the south-east, however, the ground is more broken by foot-hills and valleys. In both areas tea can be grown up to the steeper slopes which extend along the base of the cliffs that form the greater part of the 6,000 to 9,900 ft. to which the mass rises above sea level. These cliffs form almost vertical scarps, 4,000 to 5,000 ft. in height, along the southern and western faces of the mountain. Away from the mountain tea is found only to a distance of a few miles towards the plain. The rainfall is the limiting factor to extension both in the length and breadth of the Mlanje tea district, except to the east where the Portuguese frontier intervenes. (Figs 1 & 2.)

Several permanent rivers and streams traverse this area from the mountain towards the plain, and in the rainy season the cliffs are seamed with dozens of waterfalls owing to the very high precipitation on the upper levels of the mountain. The Great and Little Ruo falls, the former some 800 ft. in height, carry permanent streams that unite towards the Portuguese border to form the Ruo river, one of the main rivers of southern Nyasaland.

Practically the whole of this area was within recent times clothed with virgin forest, mostly of medium or small trees but reaching a good size along the streams. In a few cases the tea gardens have been cleared from grassy slopes with scattered trees, but most of those now in bearing were till recently fairly dense forest, and this is, no doubt, the explanation of the particular types of disease most prevalent in the tea. Some of the existing gardens were originally cleared for coffee planting, but even in these cases the conditions often remained almost like those of a forest, owing to the large trees employed as shade and the growth of the coffee itself.

The soils are lateritic, varying in colour from red to grey, chocolate, or deep brown, and are weathered from the orthoclase-hornblende-syenite of Mlanje and the older metamorphic rocks, including a variety of crystalline schists and gneisses, of the Shiré Highlands. They are, in general, adequately to richly provided with the essential plant foods, the phosphorus, however, being largely in an unavailable condition. The percentage of iron and aluminium is high and the soil reaction is generally acid. The darker soils are rich in organic matter. On the slopes and ridges the soils are generally very deep and the texture is open, permitting free drainage and aeration. In the hollows ("dambos") which are a characteristic feature of the Nyasaland highlands, the clay subsoil may come near the surface and the soil is stiffer, requiring deep drainage. In these hollows root disease is, so far as I could observe, entirely absent from the tea, but I was informed that they were ordinarily devoid of trees and this, rather than soil conditions, may be the reason for their immunity from root disease.

Round Mlanje the tea flushes in the rainy season from December to April, but plucking becomes impossible when the flush is checked as the cold weather sets in. This immediately follows the rains and precedes the hot season in October to December. That crops exceeding 500 lb. to the acre can be obtained in a four months' season, during which there is also a constant battle with the weeds and grass which grow so fast that they can scarcely be held in check, is a sufficient tribute to the fertility of these soils and the forcing effect of the rainy season. Much of the rain falls in short sharp showers, succeeded by periods of bright hot sunshine, and these conditions induce an intensity in the development of weeds and in the flushing of the tea with which none but exceedingly fertile soils could cope. This fertility is natural, since manuring is by no means general: when the land is first cleared it appears to be high in both the top and the deeper layers of the soil, but after clearing there are indications that the top soil loses fertility through the action

of wash and possibly of other factors as well. In analyses of severely washed, unfertilized Mlanje soils that I have seen, where no attempt was made to separate the surface from the subsoil, the results indicate that the essentials for good tea soils are more fully provided than is common in Ceylon.

In the rest of the Shiré Highlands the rainfall is generally insufficient for tea. The area suitable for tobacco is large but the only other district in which tea is being planted is Cholo, where with a rainfall of only about 60 inches and a colder climate, crops of 400 lb. to the acre are likely to be difficult to obtain. In Mlanje, however, less than half the available land suitable for tea is at present in bearing, and extensions now contemplated should more than double the output. The yield per acre is also being increased in some gardens by fertilizing and improved soil and bush management, while in quality, at present distinctly low, there is also evidence of improvement with the growing of better "jats" (varieties) of bush and more experience in manufacture. Both yield and quality are at present low on the average and one, or preferably both, must be improved if the district is to prosper in the face of rising labour charges. The labour is mainly migratory, and is short from December to February but generally sufficient for the rest of the year. The shortage in the early part of the rains is the chief reason for the frequent failure to keep the weeds down during this period, in which the bushes may become almost hidden from sight in the long grass and other vegetation.

Considering the physical and chemical properties of the soil, the root development of the tea is somewhat disappointing. This is particularly noticeable in young bushes, one to two years after planting out. In such bushes it is common to find no lateral root development in the first eight or ten inches of soil. Small laterals form but fail to provide a root system. Taproot development, however, is vigorous and lateral systems of considerable size early appear in the deeper layers. Subsequently, a surface lateral system arises below the collar of the bush, no doubt as a result of the vigour imparted to the plant by the deeper systems. The mature bush has frequently a substantial growth of stout laterals a few inches below the surface of the soil, the taproot continuing to show strong laterals as it is followed down, but in many cases the young bushes have no hold in the surface soil and if the taproot is broken at a depth of eight or ten inches the bush comes away with a slight pull. Even in mature bushes there is often a lack of surface feeding roots while a tendency for the stout surface laterals to turn down towards the deeper layers has also been noticed.

In endeavouring to account for this type of root development, which must reflect on the vigour and health of the bushes, attention was early attracted to the prevalence of soil erosion and surface wash. It was quite evident that many of the slopes had suffered severely in the past from the loss of the surface soil by rain washing. A rainfall of two inches in an hour is said to be not infrequent in the early part of the rainy season, and a fall of nearly nine inches in 24 hours occurred during my visit. Under such conditions the loss of surface soil must be very severe when the protection of the forest is removed. The loss will vary with the steepness of the slope (rate of flow of the water) and the nature of the soil. The organic matter and fine particles, of most importance to fertility, are the most readily affected by wash and with their loss there is a reduction in the water-holding capacity of the soil. The first heavy rains after the hot season are likely to be the most destructive: later on the growth of weeds, and drainage by the establishment of percolation to the lower layers of the soil may diminish the damage. During the hot dry weather, however, the weeds die down, and the less deep cultivation the soil gets the greater the run-off on the first rains and the resulting loss of fertility in the upper layers. The Nyasaland Agricultural Department called attention to the evils of soil erosion in the strongest terms in Bulletin No. 1 of 1924, in which Captain A. J. Hornby, the Agricultural Chemist to the Department, stated that "tree growth has been destroyed for native gardens down to the banks of streams, and with no precautions we may say that the run-off has risen from 33 per cent. in the forested areas to 69 per cent. of the annual rainfall on such lands. The agricultural capital has been allowed to run to waste and can never be replaced by any system of manuring." These warnings, together with the advent of planters with recent Indian and Ceylon experience, led to a serious effort being made in some tea gardens to combat erosion. In a great many places, however, the damage had already progressed very far, and in some, new areas are still being cleared without any provision for checking wash. In these last cases the top soil constituents of value for the feeding purposes of the tea plant are being removed faster than they are formed and a quite obvious diminution in fertility is taking place. That this, in itself, is capable of influencing the progress of root diseases is indicated by the fact that in certain gardens where no effort has been made to combat disease directly but where wash has not, for one reason or another, been severe and manuring has been practised to some extent, the patches of root rot, though numerous, are mostly

of small extent and slow spread. In these gardens the susceptibility to prevailing root diseases is clearly less than where wash has been severe and soil fertility has been allowed to decline.

Field observations indicate that the types of root disease most commonly found in the Mlanje tea are those which are most easily able to affect bushes with a relatively weak root development. Other root fungi of types which are indifferent to the vigour of their victims (well-known examples of which in other crops are the *Fusarium* wilts) do not appear to occur. The poor development of the bushes on severely washed slopes, as evinced by the absence of spreading growth, the yellow colour of the leaves, and weakness of flushing is accompanied in most cases by outbreaks of disease and is, in my opinion, one of the two main factors which have assisted in causing the unusual prevalence of root diseases in this area. Whether there are other minor factors, working in the same direction as that just discussed in weakening root development, is uncertain. It is possible that the apparent infertility of the upper soil layers may be in part due to the heavy falls of rain forcing the soluble plant foods down to the lower levels. It is also possible that the dry season is sufficiently prolonged to cause drought conditions in these layers, so that the early formed laterals cannot obtain sufficient moisture for their development. Weeds may also play a part by enfeebling the growth of the green parts of the bush and thus diminishing the plant food required for forming a strong root system. Observations carried on for a more prolonged period would be necessary to determine these points.

The second main factor predisposing to root diseases in the Mlanje tea is the wide-spread failure to "stump" the ground when it was first cleared from forest. Only in a few cases were the stumps and main roots of the forest trees removed: in most gardens they were left to decay in the soil, with the same result that has been experienced in all tropical and sub-tropical countries when this precaution has been neglected. As the stumps decay, patches of dying tea are noticed around them, and these may continue to expand for a considerable period unless arrested. I have not seen an adequate explanation of this phenomenon, though the immediate cause of the death of the bushes the action of certain parasitic fungi which attack the root is well known. How it is that these fungi require the presence of decaying wood, usually in large masses, to start them into activity is obscure. As in other countries, so in Nyasaland, it has been noticed that certain trees are more apt to start root disease than others. The planters generally were agreed that the two worst trees in this respect were the "Mwanga" (*Afrormosia angolensis*) and the "Muula" (*Parinarium mobola*), the former a very hard wood and the latter moderately soft. As also in India and Ceylon, there is no evidence that the trees most implicated in this trouble are related to one another either botanically or in their physical characters. *Grevillea robusta*, which is found in some numbers as a shade tree in the tea, has not yet reached an age when its well-known tendency to start root diseases would show up: observations in other countries show that the tree is harmless so long as it is growing, and only becomes a menace after it is cut down and the stump left to rot in the ground. *Albizzia moluccana*, another known offender, is occasionally used as shade and will, no doubt, prove as troublesome as it has in Ceylon. The indigenous *A. fastigiata* ("Chikwani") is suspect but has not been proved to cause trouble. Others, such as a local *Ficus*, were mentioned to me as dangerous, while the "Mbawa" or African mahogony (*Khaya senegalensis* var. *nyasica*) belongs to a genus that is known to be susceptible to one of the root rots (*Armillaria mellea*) prevalent in Nyasaland, but whether it can initiate an attack is uncertain. It is obvious that definite information on these points could only be obtained during a longer period of observation than was available to me.

Enquiries led to the conclusion that the root disease patches begin to appear usually about five to seven years after the forest is cleared, and they may persist, if unchecked, for many years. Mwanga stumps were found still fairly sound after 15 years, and there is no reason to doubt that such stumps could initiate delayed outbreaks, just as in India and Ceylon attacks are known to have started from stumps 14 years old.

Of the four fungi hitherto identified as causing root diseases of tea in Nyasaland, two, namely, *Armillaria mellea* and *Ustulina zonata*, are forest fungi. The former is known to be able to attack potatoes, but the disease thus caused is exceedingly rare and, in general, it may be said that annual plants growing in arable soils are free from both species. What may be called forest conditions - shade, undisturbed soil, plentiful supplies of organic matter, high soil moisture, and the like—further their development. So far as I am aware they are generally found in other countries only to a moderate depth in the soil and their presence below two feet appears to be rare. In the loose, well-aerated Mlanje soils, however, they go deeper, at least to two and a half feet. Moisture is essential to their growth and, leaving aside the hollows known as "dambos" already mentioned, they appear to be more prevalent the moister the soil. The low evaporation from, and high moisture

capacity of forest soils are, therefore, particularly favourable to these two fungi. At first sight it might appear that the soil conditions on the washed slopes in which root diseases are most prevalent are not such as would favour these fungi. They undoubtedly persist, however, on the rotting timber in these soils, the absence of drainage and the poor cultivation help their development, and the weak root system in the top soil renders the bushes particularly susceptible to their attack.

The other two, *Botryodiplodia theobromæ* and *Macrophomina phaseoli*, are found in arable and grass as well as forest soils and are known to attack annual plants. The conditions favourable to their development are clearly not the same as with the first two, and there is little evidence that they start from rotting stumps.

Besides the diseases caused by these four fungi another, and in some respects the most serious, of the tea diseases encountered in Mlanje has many of the characters of a root disease though no parasite has been found in the roots of affected bushes. Its occurrence will be more fully discussed below.

Throughout the whole of Mlanje tea district these five diseases occur probably in every garden. Stem and leaf diseases also are found but, except for the stem canker associated with *Macrophoma theæ*, are of little importance. In particular the two serious diseases red rust, caused by *Cephaleuros parasiticus*, and thread blight, caused by several species chiefly of the genera *Corticium* and *Marasmius*, are unknown. The harmless leaf form of red rust (probably due to the allied *C. mycoidea*) is not uncommon; but a careful search failed to disclose any but a few small superficial patches on the stem, usually just below the insertion of the leaves. Of the leaf diseases, the grey and brown blights (*Pestalozzia theæ* and *Glomerella cingulata*) occur, as they do wherever tea is grown, but the damage done by them was negligible during the period of my visit, though I was informed that they had been severe in places in 1926, possibly as a concomitant of the rather severe infestation by mosquito blight (*Helopeltis bergrothi*) in that year. Even in the oldest tea seen (there are a few areas with bushes over 30 years old) the above-ground parts of the plant were remarkably clean and free from blights, possibly as a result of the relatively long dry period in the climate of the district. Of the serious root diseases of tea known in other countries, those due to *Rosellinia* spp., *Fomes lamaœnsis*, and *Poria hypolateritia* were not encountered.

In the tea seedlings still in the seed-beds *Macrophomina phaseoli* was associated with a rot at about the level of the seed, and heat canker was found in a few cases. In young tea (one to two years after planting out) *Botryodiplodia theobromæ* and *Ustulina zonata* caused more deaths than either *Armillaria mellea* or *Macrophomina phaseoli*. The disease of unknown causation may also have been responsible for some of the losses in young tea, but its incidence at this stage was not easy to determine. *Macrophoma theæ* was reported to have been destructive in seed-beds and young tea in 1926, but owing to the vigorous control measures adopted was only prevalent during my visit in a few areas of planted-out tea that had not yet been pruned. Spreading patches of root disease were not observed in young tea. In the mature gardens *Armillaria mellea* and the obscure disease were far more frequent than the others. *Macrophomina phaseoli* was infrequent on full-grown bushes and was only once found on the roots of a dead bush unaccompanied by any other pathogenic fungus, though somewhat more common on single dead roots, in one case accompanied by death of a few branches above.

A more detailed account of these various diseases, the only ones of importance observed, follows.

Root and Collar Crack Caused by Armillaria mellea.

The tea disease caused by *Armillaria mellea* has previously only been reported from Java,[1] where it is found mostly at altitudes of 4,000 ft. or over, *Cinchona* also being attacked, and from Uganda,[2] where it is said to be the commonest cause of root disease in a considerable number of woody plants, amongst which tea is mentioned. The fungus itself, a well-known species in temperate zones, is not common in the eastern tropics; it has not been seen in Ceylon, while in India it has only been recorded at rather high altitudes (over 7,000 ft.) in the Himalayan pine forests. In Africa, however, it is more frequent: in the Gold Coast it is the cause of a serious disease of cacao (*Theobroma cacao*) to which the name "collar crack[3]" has been given, and has also been found attacking the African mahogany (*Khaya* spp.), mango (*Mangifera indica*), avocado pear, *Cola acuminata*, *Chlorophora excelsa*, *Funtumia africana*, coco-nut, the oil palm (*Elaeis*

1 Palm, B., "Over de door schimmels veroorzaakte wortelziekten van de theeplant. III. Beschrijning van eenige wortelziekten."—Meded. Proefstat. voor Thee, LXI, 1918.

2 Small, W., "The diseases of Coffea arabica in Uganda."—Uganda Dept. of Agric. Circ. 9, p. 20, 1923.

3 Dade, H. A., "Collar crack of cacao (*Armillaria mellea* (Vahl.) Fr.)"—Gold Coast Dept. of Agric. Bull. 5, 1927.

Fig. 3. -Collar of old tea bush killed by *Armillaria mellea*, showing cracking and white mycelial sheets.

Fig. 5.—Root of tea bush killed by *Armillaria mellea*, showing longitudinal bands of xylostroma and (above) white mycelial layer in the bark.

Fig. 4.—Taproot of tea bush killed by *Armillaria mellea*, showing black frills of xylostroma and (below) black lines in the wood.

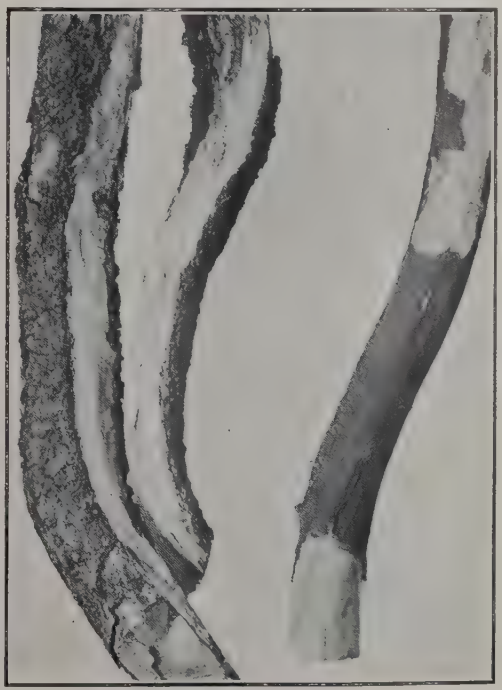

Fig. 6.—Roots of tea bushes killed by *Armillaria mellea*. Left, root split into two pieces, the inner faces of which are lined with white mycelium. Right, below, black line in wood continued as xylostromatic band on surface; above, continuous white mycelial sheet in the bark.

guineensis), and other trees and herbaceous plants, while in north Africa it causes a root disease of fig trees. In Nyasaland Mr. J. B. Clements, Conservator of Forests, has reported it[1] on the Mianje cedar (*Widdringtonia whytei*), the successful planting of which on Zomba Mountain has been seriously interfered with on account of its ravages, and I saw mangoes, *Cedrela toona*, and *Poinciana regia* dying from what appeared to be the same disease, while the brown collar rot of coffee observed in the Ncheu district is possibly due to this fungus.

On tea it causes a wet rot affecting the tap root and main laterals, sometimes also extending into the stem well above soil level (fig. 3). The rot is so wet that water can be wrung out of the affected roots, which in late stages of the disease become softened and pulpy. This wet rot is also frequently found in cacao affected with collar crack and has been ascribed to the action of secondary bacterial invasion. The chief symptom on the affected parts is the presence of conspicuous, raised black lines or narrow bands running longitudinally on their surface (figs. 4, 5, 6), and usually representing the external growth of radial sheets of mycelium (xylostroma) which ultimately disrupt the wood in the direction of the medullary rays and lead to radial cracks separating the affected root or stem into elongated, wedge-shaped strips. In the early stages the woody tissues remain firm, but it soon becomes possible to tear the root longitudinally into two or more strips, the inner faces of which are lined by white or dingy mycelial sheets (fig. 6). At a later stage the whole of the wood softens and rots. The cortex is early penetrated by sheets and strands of white mycelium, running chiefly parallel to the surface of the bark and producing a mottled appearance, due to alternations of white mycelium and brown bark, when the latter is shaved away tangentially. On careful dissection the sheets are found to form a more or less continuous layer (figs. 3, 5, 6), which, when growth is active, extends either through the middle of the bark or just external to the cambium for a considerable distance, the growing margins being spread out fan-wise. These symptoms are very similar to those described on cacao in the Gold Coast and they exactly resemble those shown in the photographs of the split-canker of tea in Java caused by *A. mellea*.[2] Rhizomorphs are occasionally found on the surface of the affected roots as black, rounded or flattened strands, closely resembling black roots, a couple of millimeters or more in diameter. They are rare under African conditions and were only found on one tea bush in Nyasaland. This bush, which was nearly dead from a typical attack, also bore clusters of sporophores, medium sized toadstools, in its centre, and other similar clusters were found around the base of a dead *Poinciana regia* tree bordering the tea, in a line in which several deaths had occurred from root disease. Sporophores are evidently formed much less often in tropical Africa than in Europe and they are generally smaller and more deeply coloured and the fungus forms rhizomorphs less regularly than in the temperate forms of *Armillaria mellea* so familiar in woods in England.[3] In Java the form which causes the white root rot of *Cinchona* has been grown in pure cultures[4] in which it produced toadstools in clusters of two or more, yellowish-brown to brown, the stalks 5 to 6 cm. high by 5 to 6 mm. broad and caps 5 to 6 cm. across. No ring is present in the mature specimens of this form, but one can be seen when they are young. The spores are hyaline, ovoid-elliptical or elliptical and slightly pointed, measuring 7 to 9 by 5 to 6 microns, and borne on whitish gills which touch the stem and are somewhat decurrent. Rhizomorphs are not abundant but form readily in culture. Still smaller forms occur in the Java mountains and in the Cameroons and have been recorded as *A. mellea* var. *javanica* and var. *camerunensis*, respectively:[5] the first is described as lurid brown, darker at the top, 2 to 3 cm. in diameter, with a hollow stalk up to 5 cm. high and 2 to 3 mm. thick, a white ring, adnate gills, and hyaline, subglobose spores, 6 to 8 microns in diameter, while the second was only $\frac{1}{2}$ to 1 cm. across. The Nyasaland specimens were too old for accurate description but they would pass for old *A. mellea*, and the presence of rhizomorphs and general characters of the disease leave no doubt as to its causation by this fungus.

The most severe attack of *Armillaria mellea* observed was in a garden planted in 1915 on land cleared from forest containing a considerable number of trees of *Parinarium mobola* and

1 Clements, J. B., "Report of the Chief Forest Officer."—Ann. Rept. Nyasaland Dept. of Agric. 1925, p. 19, 1926.

2 Bernard, C., "Aanvullende mededeelingen over de wortelziekten van de thee."—Meded. Proefstat. voor Thee, LXIa, Pl. III, 1919.

3 Gard has recently described ("Rev. Path. Vég. et Ent. Agric.," XIII, p. 183, 1926) a form of this fungus attacking the walnut in France which seems so have many points in common with the tropical African form: In the United States the fungus known as *Clitocybe tabescens*, which has been regarded by some as only a variety of *A. mellea*, causes a root rot of vines and of fruit and forest trees which closely resembles that here described.

4 Rant, A., "The white root-fungus of Cinchona."—Recueil des Trav. Bot. Neerlandais, XIV, pp. 143-148, 1 pl., 1 fig., 1917.

5 Hennings, P., "Fungi monsunenses."—*Monsunia*, *I*, p. 20, 1899: *ibid.* "Fungi camerunenses, I."—*Engler's Jahrb.*, XXII, p. 107, 1895.

Afrormosia angolensis. Some ten acres were affected and 75 per cent. of the original bushes had been killed. The field sloped down to a deeply-drained valley at right angles to the forest which bounded it on one side, and was separated by a road on the side opposite the latter from another garden of the same date. A great part of the ridge and slope had bare patches and young supplies, but the bottom of the slope was without a single case of disease and the latter was less severe on the side furthest from the forest, though patches reached the road in several places. That the road had checked extension considerably but not entirely prevented it was evident from the fewness and small size of the patches in the part of the second garden adjoining it. Out of some 20 bushes examined, only three were without symptoms of *Armillaria* attack in the top foot or so of the root system and these possibly were affected lower down. The bushes died gradually and generally from one side, this unilateral type of attack being characteristic of most of those caused by *Armillaria* that were seen. The garden had not been stumped after clearing from the forest, there was no shade, weeds were few, and wash had been unchecked and was doubtless considerable.

A few miles away, in another estate, several gardens of heavily shaded old tea were seen in which there were numerous patches of root disease of the same type, but these patches were small, isolated from one another, and evidently spreading slowly. The soil had been fertilized with cattle manure and a growth of low weeds encouraged with a view to checking wash. In this estate, where the tea bounded the still-standing forest, its edges were scalloped with numerous semi-circular patches of larger size than those in other parts, the appearance strongly suggesting that the attack was extending into the tea from the living forest. I was informed by Mr. Clements that this phenomenon was very apparent in his Mlanje cedar plantations and that he had checked it by trenching. The relatively low percentage of loss in these last-mentioned gardens may be attributed to the vigour of the bushes resulting from fertilizing and the prevention of wash, as stumping had not been done. Where extension was going on, the attack on the bushes was nearly always unilateral, half the bush often being dead while the rest was more or less normal.

Similar small, sharply-defined patches were seen in another estate in gardens cleared from very heavy forest in 1911-12 and stumped after clearing. Some of the old *Afrormosia* stumps were still undecayed. The attack was generally unilateral and every case examined had the typical root cracking. It is evident that neither stumping nor fertilizing will prevent the attack of *Armillaria mellea*, but the small patches in the two last-mentioned estates should be easy to control.

Apart from these spreading patches (various intermediate types of which between the small, sharply defined patches and the larger, irregular ones often intercommunicating, were seen), a number of isolated deaths from the same type of root rot were met with. The attacks killed the bush very rapidly, causing it to wither up completely with its leaves still attached. These single withered bushes with brown or even still green dead leaves are very conspicuous. In one place three of them were found near together and a patch was probably starting, but wherever good sized patches were established, the deaths at the margin of the patch were gradual and preceded by the shedding of the leaves. Even in these gradual deaths it is rare to find new shoots arising from below the diseased branches, though new ones may continue to appear from the sound parts of the bush. In one or two cases, however, a few healthy surface lateral roots from which healthy shoots arose were found in bushes of which the rest of the root system and above-ground parts were dead. In the only instance in which this disease was found in young tea, the bush died in its second year after planting out and its taproot was marked by the characteristic black lines on the surface and mycelial sheets in the bark.

Microscopic examination shows that the mycelium penetrates all the tissues of the affected roots and collar, the hyphæ passing from cell to cell either through the pits in the cell walls, or by boring through the latter. Three types of mycelium are found, a colourless feeding mycelium of slender, branching, hyaline hyphæ; a brown stromatic mycelium confined to narrow layers that appear as black lines when the wood is cut; and a white or yellowish aggregated mycelium forming the sheets in the cortex and in the cracks in the wood, sometimes coming to the surface, where the free edge turns black and produces the thick black bands on the surface of the affected parts. These last are a transitional form to the true rhizomorphs, which are free, thick, leathery, black strands, sometimes flattened and resembling bootlaces, sometimes rounded and like roots spreading through the soil.

The exact manner of infection was not determined, beyond that it was associated in the early stages with the presence of rotting stumps or buried timber in the soil. In temperate countries infection occurs from rhizomorphs which spread through the upper layers of the

soil and infect healthy roots with which they come in contact.[1] In Uganda also spread by rhizomorphs is stated to have been observed,[2] tea bushes having become infected by rhizomorphs which had passed over from a diseased ceara rubber tree (*Manihot glaziovii*). In the Gold Coast spread occurs along the roots, and infection is due to direct root contact. In Nyasaland it is probable that, even though rhizomorphs are rare, spread occurs through the soil, the frequently even centrifugal spread being difficult to explain in any other manner, since the larger tea roots are not often in contact.

Infected roots always show the white sheets of mycelium in and under the bark, and the formation of these appears to take place at an early stage. Underneath them the wood is permeated by hyphæ. Under conditions that are not understood these hyphæ tend in certain places to form swollen bladder-like cells completely filling the cell cavities of the host. This formation starts in the medullary rays and it is possible to find stages in which it is confined to them. More usually, however, it occurs in a continuous layer, reaching from near the surface of the wood to a variable depth towards the centre, and involving all the cell elements, tracheids, fibres, and wood parenchyma. When the band, as is frequently the case, extends along the natural grain of the wood of the root, splitting occurs owing to the destruction of the cell walls, of which frequently nothing but the spiral or scalariform thickenings are left. The cracks thus formed are usually but by no means invariably radial, tangential cracks being not uncommon (fig. 6). When the band crosses the natural grain it does not lead to splitting. The stages of the development of this bladder-like mycelium are accurately described by Hartig,[3] but it was not accompanied, in the trees observed by him, by cracking of the wood. The latter phenomenon, indeed, appears to be rare in the forms of attack seen in temperate countries.

The bladder-like mycelium is at first colourless and thin walled, but soon turns light brown and the walls thicken. In advanced stages the band, seen in section as a narrow black line, consists of a dense mass of angular, often rather small, cells, of a deep brown colour, completely occluding the cavities of the invaded host cells. When cracking occurs or the cell destruction gives room for further development, an extension of fungal growth takes place in the form of masses of elongated, closely appressed cells, which develop into sheets filling the space. Even before any crack is visible these sheets may be found on microscopical examination starting from the stromatic layers of bladder cells. The sheets are much less highly coloured than the bladder-mycelium and may even be almost white, and they are fringed by a silky outgrowth of very fine hyphæ, the lumen of which is almost or quite occluded. At the surface they turn black and thicken up into almost cylindrical masses or protrude from the crack in the form of a black frill. A similar formation can occur in the cortex, typical black bands being sometimes found arising from the white sheets in the bark when the wood below shows no sign of splitting or even of the black lines that precede the formation of a crack (fig. 6, top of root on the right).

In the accounts of black-line formation by this fungus that have previously been given[4] it is stated that the lines move gradually forward as the rot progresses into the tissues, marking the limit between two different stages of the decay of the latter. As new host cells are filled with the bladder-mycelium in the direction in which the rot is advancing, those behind become emptied by the bleaching and dissolution of the fungal cells. While it is true that the line divides the wood into areas that differ in appearance and in the extent of the decay that has been caused, no evidence was seen that its position progressively shifts forward. It appears to be laid down in the position that it continues to occupy definitively and the subsequent changes that occur in the same position often lead to the formation of a crack. The whole process is much more like the development of some defensive reaction on the part of the host or the parasite.

The measures to be adopted for the control of this disease will be given after that next to be described, as they are generally similar in the two cases.

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1 Day, W. R., "Parasitism of *Armillaria mellea* in relation to conifers."—Quart. Jour. of Forestry, XXI, pp. 9-21, 1927.

2 Small, W., *loc. cit.*

3 Hartig, R., "Die Zersetzungserscheinungen des Holzes," pp. 59-62, Pl. XI, figs. 1-5, 1878.

4 Hartig, R., *loc. cit.* Hiley, W. E., "The fungal diseases of the common larch"—Oxford, Clarendon Press, 1919, pp. 155-158.

Stump Rot caused by Ustulina zonata.

Of the several forms of stump rot, that is to say diseases arising from rotting stumps in the soil, that caused by *Ustulina zonata* is the commonest on tea in Ceylon and probably also in India.[1] The parasite is widespread throughout the tropics, attacking a number of woody plants, the most important of which is Pará rubber *(Hevea brasiliensis)*. It frequently infects coffee, tea, rubber, and the like on old forest land in Kenya, where even temperate fruit trees, such as the peach and pear, have been killed by it. It is also known to occur in Uganda and the Gold Coast but does not seem to have caused serious losses in either case. In Java, where it is known as the root-collar disease,[2] a few serious outbreaks have been recorded, but as a general rule *Ustulina zonata* is not regarded as a serious trouble in tea in that country. The fullest studies of its parasitic action have been made on rubber,[3] and in this host conclusive proof of its parasitism has been obtained by inoculation experiments.

No certain cases of root disease of mature tea associated with the presence of this fungus alone were observed in Mlanje. Only in diseased young bushes was *Ustulina zonata* found alone, though it must be presumed from experience elsewere that it is capable by itself of killing the mature bushes. I also failed to find its fructifications on tea, though these are usually fairly frequent where the disease due to it is present, and the diagnosis is, therefore, open to some doubt. Ripe fructifications were, however, obtained from just above ground level on the trunk of a *Poinciana regia* tree bordering the tea and suffering from the root disease typical of this fungus, which had resulted in a weakening of the root system and led to the tree being blown down by the wind. They were also collected in a similar situation on an unknown forest tree that had been removed from the tea in another estate.

In a typical attack of *Ustulina zonata* the tea dies out in spreading patches, which in Ceylon usually start from the stumps of *Grevillea robusta* or *Albizzia moluccana*. The parasite spreads chiefly below ground, though cases have been reported of aerial infection through its spores reaching wounded surfaces, such as pruning cuts, in India and Ceylon, and similar wound infections are not uncommon on rubber in Malaya. In the soil, spread is only possible when an infected root touches or lies very near a healthy one, the mycelium, unlike that of *Armillaria mellea*, extending only along the roots and not through the soil. In some cases in Ceylon a line of dead bushes has been found to follow the course of a main lateral root of *Grevillea* or *Albizzia*. The bushes may be killed rather gradually, losing their leaves little by little, or quite suddenly with most of the leaves attached and either brown or still greenish. In spreading patches the attack is often unilateral, part of the bush dying while the rest appears almost normal. As in the case of bushes attacked by *Armillaria*, it is rare to find suckers or new shoots arising from below the killed branches.

The affected roots bear no external mycelium except black lumps or crusts, in this resembling *Armillaria* where, however, the external crusts are arranged in narrow longitudinal bands. In the bark, too, the symptoms of the two diseases are somewhat similar, except that the sheets are smaller, more fan-shaped, and creamy-white in *Ustulina* instead of being in large uniform layers and often pure white, as in *Armillaria*. There is no tendency to cracking of the wood, but the latter is marked with sharp, angular or wavy black lines, very similar to those caused by *A. mellea* both in appearance and in the manner of their formation.

The absence of longitudinal black bands or frills on the surface of the bark and of radial cracking of the affected wood enables this disease to be readily distinguished from that due to *Armillaria*. It was never found by itself in Nyasaland causing spreading patches of disease, and in every case seen where single mature bushes died without shedding their leaves, *A. mellea* was present and seemed to be the cause of death. It is, therefore, uncertain whether *U. zonata* is a primary cause of disease of mature tea in Nyasaland, though the knowledge of its presence in young tea and in the forest makes this highly probable.

In young one- to two-year-old tea *Ustulina zonata* was sporadic and usually associated with *Botryodiplodia theobromae*, an association which was not found in older bushes. Occasionally it seemed to occur alone, the attack then being near the soil level. These isolated attacks in young

1 Petch, T., "Diseases of the tea bush."—Macmillan, 1923, pp. 141-146. Tunstall, A. C., "Tea roots, II."—Indian Tea Assoc. Sci. Dept. Publ. 44, pp. 11-12, 1918. Butler, E. J., "Fungi and disease in plants."—Calcutta: Thacker, Spink and Co., 1918, pp. 438-440.

2 Bernard, C., and Steinmann, A., "De wortelkraagziekte."—*De Thee*, V, pp. 21-23, 1924.

3 Sharples, A., "Ustulina zonata—a fungus affecting Hevea brasiliensis."—F. M. S. Dept. of Agric. Bull. 25, 1916.

tea near the surface of the soil can scarcely have been due to spread from previously diseased roots but had much more the appearance of wound infection from spores, probably associated with injuries during cultivation.

The fungus at first penetrates the bark and reaches the junction of the bark and wood where it spreads out to form delicate creamy-white fans. It then penetrates through the medullary rays into the wood, where the same two types of mycelium are found as in *Armillaria*. The bladder cells forming the black lines do not, however, cause any splitting of the wood and do not grow out to form sheets or black xylostroma. The only part that reaches the surface of the root is ordinarily the fructifications, which arise on the collar and larger diseased roots when these are above soil level. The mycelium in these situations spreads out on the surface in small yellowish-white plates, lying close to the bark but only attached at one point. Frequently several plates unite by their margins to form fairly extensive, corrugated sheets, several inches across. The single fructification is zoned, and when mature is finely punctate with black dots which mark the mouths of the spore receptacles. Before this stage, however, when the surface of the plate is still whitish and soft, very small spores (conidia) develop in a continuous layer, covering the plate, and as these have been shown to be able to give rise to cultures of the fungus they must play a part in disseminating the disease. The perfect stage arises later, when the crust is dark brown or blackish-purple and brittle, in the form of a layer of flask-shaped spore receptacles (perithecia) buried just below the surface of the crust, only their mouths projecting to form the little black dots referred to above. In these receptacles a second spore form is developed consisting of large, opaque black or dark brown, boat-shaped ascospores, arranged in groups of eight in thin cells or asci. This is the only stage observed in Nyasaland and it is probable that most of the cases of this disease observed in the young tea came from infection by means of these ascospores, which were abundant in the specimens collected on forest and shade trees. Further investigations may show that the typical spreading patches so commonly caused by this fungus in Ceylon around *Grevillea* stumps also occur in Mlanje and, therefore, the control measures described below are designed to meet this possibility.

Control Measures against Armillaria and Ustulina.

Both the root diseases caused by *Armillaria mellea* and *Ustulina zonata* are amenable to a considerable measure of control on somewhat similar lines, especially if steps to prevent their appearance in new clearings are adopted. For this purpose it is scarcely necessary, in the restricted Mlanje area, to attempt to distinguish one from another. The main points to be borne in mind are that both are favoured in their earlier stages by the presence of rotting wood in large masses in the soil and that their spread is chiefly underground.

It is not safe to clear forest land for tea planting in the Mlanje area without stumping. The two fungi are present throughout the district and no estate is free from them. Both seem to be equally capable of starting from rotting tree stumps in the soil, for in no other way can the isolated spreading patches of disease round such stumps be explained. *Armillaria* is evidently also able to extend from the still standing forest into adjoining tea, and *Ustulina* is liable to infect pruning cuts and other wounds by spores, though this is not common except, perhaps, in young tea. The value of stumping is recognized in the district, and not only is most of the newly cleared land being stumped but buried stumps are being dug out in some gardens from the planted-out tea. It is obviously better to remove them before the tea is planted, and this is the first and most essential measure in attempting to secure freedom from root disease. Fortunately, most of the forest consists of small trees and it is only in certain places that large *Khaya* and other trees are found. Some of these might, perhaps, better be dealt with by isolating them by a trench than by digging them out, the large surface laterals being cut through and removed.

In spite of these precautions root disease may appear and a careful watch must be kept to detect any attacks in an early stage. As soon as a bush dies it should be removed and adjoining bushes examined to see if they show signs of attack. Very often several bushes near together are affected almost simultaneously. When this occurs or when a definite patch has appeared, trenching to prevent the underground spread of the fungi should be at once resorted to. No other method can replace trenching as a control measure in small patches. The trench should be three feet deep and should surround the patch and one row of apparently healthy bushes, for it is obvious that the latter may be so recently infected as to show no symptoms above ground, and failure to isolate them will make the trenching useless. The soil from the trench should be thrown into the patch, not outside. Then the dead bushes should be removed, the apparently healthy ones examined on the side towards the patch and also removed if signs of infection are seen, and the

whole patch dug over to a depth of at least two and a-half feet, all fragments of roots being removed as far as possible. The bushes and pieces of root should be piled and burnt in the patch, or, if too damp to burn, should be scorched with straw or dried prunings and left until dry enough to burn. If the removal of diseased bushes is done after plucking has finished, say, in April, a second digging over should be done about October. If the work is carefully done it may be safe to supply in the following rains, say in January or February, using large, strongly-rooted seedlings for the purpose. If experience shows that such supplies are liable to contract infection it will be necessary to postpone supplying until the following rains. Supplying earlier than nine or ten months after removing infected roots is unsafe, and tests may show that even this period is insufficient to rid the soil of infection. When a single bush dies, trenching is unnecessary if the soil be well dug over after the bush is removed, and the roots of bushes in contact with it are examined. In this case a great deal will depend on whether the bush can be removed immediately it shows clear signs of root disease. If labour requirements force it to be left for some months, neighbouring bushes are likely to be infected and must also be removed. In all cases an attempt should be made to discover whether the attack has come from a rotting tree stump, and this should be removed when located.

When the disease has progressed unchecked for a number of years the patches become so irregular and extensive that trenching may be impracticable. More than one affected garden was seen in which it would be almost impossible to devise any system of trenches that would effectively preserve the still healthy bushes. In such cases reliance must be placed on the removal of affected bushes and thorough digging of the soil so as to leave as few roots in it as possible.

Liming the soil after removing the bushes is usually recommended, its main object being to promote the rapid decomposition of fragments of roots in which the fungus may be living. Where lime is cheap and easily available, so that heavy doses can be given, it is worth using, but it cannot replace thorough cultivation, whereas the latter can replace liming. There is little doubt that the more thorough and deeper the cultivation the less favourable the soil becomes for the two fungi under consideration, and the thorough turning over of the soil two or three times after removing infected roots should be effective in freeing it from them.

When the tea abuts on the forest a deep trench should be dug separating one from the other, in order to prevent continual reinfection from the latter by *Armillaria mellea*. A watch should also be kept on shade and avenue trees, and all cases of death from root disease treated in the same way as in the tea itself. It is exceedingly unwise to pile the stumps of uprooted or fallen trees in the neighbourhood of the tea as is sometimes done, owing to the tendency for ripe fructifications of *Ustulina zonata* to develop on them. If they cannot be removed they should be scorched, since fructifications were not found on the stems of scorched trees.

The above are the chief direct methods of fighting the root diseases caused by these two fungi. They are based on the fact that buried stumps and diseased tea roots are the common sources of infection. Land that is habitually kept free from decaying wood, such as ordinary plough land, does not usually contain these fungi. Trenching would be unnecessary could all diseased roots and other decaying wood be removed to a depth of say three feet, and it is being discouraged in Assam in favour of the latter procedure. But it is not always convenient to provide the necessary labour for thorough digging over when the first cases are seen, and the isolation of the patch by trenching after removing the larger roots may prevent undue spread until the ground can be thoroughly dug over. Indirect methods of prevention calculated to improve the vigour of the bushes so as to enable them to resist attack will be dealt with later, as they are applicable to all the types of root disease encountered.

Internal Root Disease caused by Botryodiplodia theobromæ.

The fungus for which the name *Botryodiplodia theobromæ* is still used, though it is extremely doubtful whether it is the correct one, is a common species in the tropics, where it is found in forest and grass land as well as in arable soils. Its parasitism has been proved in the case of certain host plants, such as cacao, sugar-cane,[1] and *Hevea* rubber[2] and there is little doubt that it can also attack tea, on which the disease attributed to it is known as internal root disease in

1 Howard, A., "On Diplodia cacaoicola P. Henn., a parasitic fungus on sugar-cane and cacao."—*Ann. of Bot., XV.* pp. 683-701, 1901.

2 Bancroft, K., "The die-back fungus of Para rubber and of cacao (*Thyridaria tarda*, n. sp.)."—F.M.S. Dept. of Agric. Bull. 9, 1911.

Ceylon and India. In many cases, however, it is found on roots and stems after they have been injured by some other agency, and its presence on dead tea roots is by no means evidence that it caused their death. In Africa it is responsible for a die back and pod disease of cacao in the Gold Coast[1] and Uganda[2] and it is associated with a root disease of apple trees in Kenya[3].

In India young tea is said to be most frequently attacked on cleared grassland and in coarse sandy soils, but elsewhere it has not been possible to associate the disease with any particular type of land. It is commonest in old tea in the low country of Ceylon, but has also been observed on young bushes and at higher altitudes. The attack usually develops only after pruning and it may increase progressively from the first cutting back given after planting out through several successive prunings until as many as 50 per cent. of the bushes are affected. Though it is not so common as the diseases caused by *Ustulina* and some other tea root parasites, it is said to be capable of killing a greater number of bushes in each individual outbreak than any other root disease.

The attack becomes visible six weeks to three months after pruning, the pruned bush sometimes failing to put out any new shoots and being rapidly killed. When new shoots grow, they may remain healthy until they are six or eight inches long and then die back. In other cases single branches may die or the bush may remain moribund for a long time. In the early stages the leaves of affected bushes or parts of bushes become mottled with pale yellow or yellowish-green patches. Then they turn black at the tips and along the edges and fall prematurely. The roots appear to be clean and healthy until they are cut, when the wood is found stained pale bluish-black. The colour is due to an internal mycelium of stout, smoky-black hyphæ which give off finer hyaline branches. There is no external mycelium and no visible accumulation of hyphæ into sheets and strands between the layers of the bark. On examining the roots with a lens, especially after death, slightly prominent, round, black bodies of small size, which are the fructifications of the fungus, may be detected almost buried in the bark. Frequently, however, they are not visible until the surface of the bark is shaved away, when they appear as small black circles with a white centre. Various other fungi found on dead tea roots in Nyasaland have a somewhat similar appearance, so that it is safer to rely on the characteristic bluish-black or slate-coloured staining of the wood for a diagnosis. The fructifications are spherical spore receptacles (pycnidia), sometimes united into clusters in a stromatic mass of black mycelium, and they contain large, oval spores which, when ripe, are uniseptate and dark brown, though they are white in the mass and unseptate when young. In the early stages the mycelium may be found only in the finer roots, but direct attack on the tap root also occurs. Later on the dark mycelium may rapidly penetrate all parts of the root system as well as the base of the stem.

A further, perfect, spore-form of this fungus has been described under the name *Thyridaria tarda*,[4] but is not generally accepted. More recently[5] it has been suggested that the perfect stage is *Physalospora rhodina*, but this has not yet been found associated with the tropical forms of the fungus.

The patches caused by this disease may be very large, and in Ceylon may develop around the stumps of *Albizzia moluccana* trees that have been ringed or cut down when they have grown too large.[6] The fungus develops on such stumps and produces spores in abundance, which infect the surrounding tea. The same tree grown as coffee shade has also proved liable to attack in Uganda.[7] Infection by spores often takes place through wounds in the larger roots caused by cultivation and also in stems as a result of pruning. Observations indicate that they usually do not succeed unless the wood has been dried out after pruning. A more frequent source of internal root disease is probably the accumulation of buried prunings in the soil, as the fungus grows and sporulates freely on such prunings and patches have been observed to start around the holes in which they had been buried.

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1 Bunting, R. H., and Dade, H. A., "Gold Coast plant diseases," p. 49.—London : Waterlow and Sons (1924). Dade, H. A.,"Economic significance of cacao pod diseases and factors determining their incidence and control."—Gold Coast Dept. of Agric. Bull. 6, 1927.

2 Snowden, J. D., "Report of the Government Botanist for 1920."—Ann. Rept. Uganda Dept. of Agric. for 1920, pp. 43-46, 1921. Small, W. "Annual Report of the Government Mycologist, *ibid.* for 1924, pp. 18-20, 1925.

3 McDonald, J., "Annual Report of the Mycologist."—Ann. Rept. Kenya Dept. of Agric. for 1924, pp. 106-111, 1925.

4 Bancroft, K., *loc. cit.*

5 Stevens, N. E., "Two species of Physalospora on citrus and other hosts."—*Mycologia*, XVIII, pp. 206-217, 1926.

6 Petch, T., *loc. cit.*, p. 152.

7 Small, W., "Annual Report of the Government Mycologist for 1921."—Ann. Rept. Uganda Dept. of Agric. for 1921, pp. 52-53, 1922.

Cases of the root disease caused by this fungus were not common in Nyasaland, except in young tea, during the period of my visit. Mature dead bushes were never found to have more than an occasional root infected, and no patches of dying bushes suffering from internal root disease were seen. The only instances in which bushes appeared to have been killed by *Botryodiplodia theobromæ* were in the first or second year after planting out, and such attacks were always sporadic. They were sometimes associated with the presence of *Ustulina*, so that it was impossible to decide which was the primary cause of death. I was informed, however, by Captain Smee, the Entomologist to the Nyasaland Department of Agriculture, that during the dry period of the year the fungus is present on the roots of many dead bushes, and it is clear that much further investigation is required before its relative importance as a cause of tea root disease in Nyasaland can be determined.

This fungus is a weak parasite, especially in the underground parts of plants, and is one of those that can best be fought by improving the general health of the bushes. There is no evidence that it is able to infect tea by the growth of mycelium from buried stumps, so that stumping has no influence on its attacks, except in so far as the wood of decaying stems and roots of such trees as *Albizzia* may serve as a reservoir from which spores are discharged into the surrounding tea. It has been noticed in India that there are few spores discharged in the earlier weeks after the cessation of the rains, and it is recommended that when heavy pruning is required in tea on areas where the disease is prevalent, it should be done during this period, and at the same time two or three ounces of nitrate of potash should be applied around each bush in order to stimulate the new growth. In such land, also, prunings should never be buried but should be collected into heaps and burnt. There is some indication in the Mlanje district that young tea is more liable to be attacked when planted on cleared grass land, as in northern India, and in such areas at least the prunings should be burnt, not buried.

The Sclerotial Root Disease caused by Macrophomina phaseoli (Rhizoctonia bataticola).

This fungus has only been recorded on tea in Ceylon, where it was first reported in August, 1926,[1] though the question of its parasitism on this host was left open pending further study. Previously it has been investigated in some detail as a parasite of various plants in India, the United States, Egypt, and Uganda, and in 1927 there arose a considerable controversy as to its importance as a cause of root disease of woody plants in Ceylon. In India[2] it attacks cotton, jute, groundnuts and cowpea and has been found on many other plants; in the United States,[3] sweet potatoes, ordinary potatoes, and beans; in Egypt,[4] cotton, cowpeas, and beans; and in Uganda,[5] some forty different plants, including coffee (on which it is reported to cause a serious root disease), cacao, eucalyptus, and a number of woody plants used as shade or green manure in tea and coffee plantations. In Kenya also it is one of the causes of coffee root disease.[6] In Ceylon the number of its hosts appears to be at least as large as in Uganda (including a high proportion of the economically valuable plants of the Colony)[7] and it is evidently one of the most nearly omnivorous parasites known. It appears to be confined to warm countries, its distribution corresponding roughly to the areas in which cotton can be grown.

In Nyasaland *Rhizoctonia bataticola* is associated with a disease of tea seedlings still in the nurseries, and it was also found sporadically on the roots of dead bushes about a year after planting out and, less often, on those of mature bushes.

The seedling disease was severe in only a few beds in one nursery, though a number of individual attacks occurred in several other nurseries involving both newly imported and acclimatized seed. The leaves of affected seedlings turn pale and then wither and fall off more or less completely. The shoot dies back to the collar, but usually new shoots arise at or below the surface

1 Small, W., "Sclerotium bataticola Taub."—*Tropical Agriculturist*, LXVII, pp. 94-5, 1926.

2 Shaw, F. J. F., "The morphology and parasitism of Rhizoctonia."—Mem. Dept. of Agric. India Bot. Ser., IV, 6, 1912. Shaw, F. J. F. and Ajrekar, S. L., "The genus Rhizoctonia in India." *ibid.* VII, 4, 1915.

3 Taubenhaus, J. J., "The black rots of the sweet potato."—*Phytopath.*, III, pp. 161-164, 1913.

4 Briton-Jones, H. R., "Mycological work in Egypt during the period 1920-1922."—Min. of Agric. Egypt, Tech. and Sci. Service Bull. 49, 1925.

5 Small, W., "A Rhizoctonia causing root disease in Uganda."—Trans. Brit. Mycol. Soc., IX, pp. 152-166, 1924. *Ibid.* "On the identity of Rhizoctonia lamellifera and Sclerotium bataticola," *ibid.* X, pp. 287-302, 1926. *Ibid.* "Notes on species of Fusarium and Sclerotium in Uganda."—Kew Bull., 1925, pp. 118-126, 1925.

6 McDonald, J., "Fungoid diseases of coffee in Kenya Colony."—Kenya Dept. of Agric. Bull. 3, 1926.

7 Small, W., "Further notes on Rhizoctonia bataticola (Taub.) Butler."—*Tropical Agriculturist*, LXVIII, pp. 73-75, 1927; LXIX, pp. 9-12, 1927.

of the ground and the plants often recover. On pulling up plants after the leaves have withered, a wet patch of partially rotted bark is found extending along the taproot and base of the stem for about half an inch at the seed level (one to two inches below the surface of the soil). This rot usually involves the seed (which may be reduced to a slimy mass probably by the action of secondary organisms), but it does not ordinarily penetrate into the woody tissues of the seedling, and in those that recover little signs of its presence are found. The lateral roots are clean and apparently quite free from this fungus. The surface of the rotting bark in all cases examined bore moderately stout, brown hyphæ of the type characteristic of *Rhizoctonia*, and the small black sclerotia that distinguish *Rhizoctonia bataticola* were found in a few roots when the bark was peeled off, though they are by no means universally present. In cotton seedlings also it is not easy to find sclerotia, and the cause of the cotton seedling disease due to this fungus, which is common in India, is often difficult to determine without cultural work. The most severely affected seed-beds were in the lowest part of a nursery, these particular beds being bounded on two sides by tall grass which kept the air moist and stagnant.

Typical sclerotia of *Rhizoctonia bataticola* were found on the taproot of two bushes which had died about a year after planting out, and from one of these a pure culture of the fungus was obtained. A very large proportion of the young tea had died in the field from which these were obtained, a field which had been recently cleared from the same belt of forest as that which gave rise to the most severe attack of *Armillaria mellea* mentioned above. In none of the other cases examined, however, could the cause of death be determined, and it is possible that the field was affected by the disease next to be described.

The only case seen in which a mature bush had apparently been killed by *Rhizoctonia bataticola* was a single dead bush surrounded by healthy tea. It had dried up completely and the tissues, especially those of the roots, were bleached, hardened, and unusually light. The collar showed no signs of fungous attack, but a considerable number of the lateral roots, from one-eighth to half an inch thick, contained numerous hyphæ and characteristic sclerotia in the inner bark and outer layers of the wood. No other parasite was found, and the death of the bush may be attributed with considerable probability to *Rhizoctonia bataticola*. In another case a bush was seen with a dead branch at one side arising from near the ground level and sclerotia of the fungus were found on two roots just below this point. On various other occasions roots of diseased bushes were found with a growth of *Rhizoctonia bataticola* usually in the neighbourhood of wounds inflicted during cultivation or otherwise, but other tea root parasites were present and may have been responsible for the diseased condition.

Tea roots affected by this fungus show little external symptoms; indeed, as a rule, none that can be detected without the use of a good lens, except the condition described above as found on the taproot of diseased seedlings. In older plants the cortex of affected roots is brittle and its inner layers may show a tendency to shredding, coming away from the wood as a bundle of fibres rather than as a single continuous sheet. In the inner layers or, more often, on the surface of the wood very small, rounded or elongated, black dots can often be seen with the lens. Microscopic examination shows that these are composed of thick-walled, brown cells forming small stromatic bodies or sclerotia. The mycelium is generally scanty and is composed of hyaline hyphæ, very variable in diameter and with the branching characteristic of *Rhizoctonia*. The older hyphæ may turn brown and at the same time show a tendency to form clusters of the dark-walled cells. The sclerotia usually appear as the tissues dry up, and the manner of their formation has been fully described by Shaw and Small. They are amongst the smallest known, averaging not much more than 100 microns in diameter.

Under certain conditions, which appear to be difficult to reproduce artificially, this fungus gives rise to a sporing form which was originally described on bean stems from Tunis under the name *Macrophoma phaseoli*.[1] This form has since been obtained on several of the hosts of the sclerotial form from Formosa, the Philippines, India, Uganda, Ceylon, and the West Indies and the most recent study,[2] in which full literature references and synonymy are given, indicates, that the correct name for the organism is *Macrophomina phaseoli* (Maubl.) Ashby. On one of the young tea bushes that had died some time after planting out, a pycnidial form agreeing fairly well with *M. phaseoli* developed near soil level in material brought to England, so that it is probable that the sporing stage of the fungus also occurs on tea. This stage on other plants

1 Maublanc, A., "Espèces nouvelles de champignons inférieurs."—Bull. Soc. Myc. de France, XXI, p. 90, 1905.

2 Ashby, S. F., "Macrophomina phaseoli (Maubl.) comb. nov. The pycnidial stage of Rhizoctonia bataticola (Taub.) Butl."—Trans. Brit. Mycol. Soc. XII, pp. 141-147, 1927.

consists of small black spore cases (pycnidia), smaller and more superficial than those of *Botryodiplodia* and containing a number of colourless, unseptate, elongated spores, measuring about 16 to 30 by 6 to 9 microns in diameter. On tea, however, the spores were only 16 to 22 by 4.5 to 6 microns, which is unusually small for *M. phaseoli*, so that it is not certain that they belonged to the *Rhizoctonia* parasite or that they play any part in the dissemination of the disease.

The accounts of the mycelial characters of this fungus are so discrepant that it is difficult to believe that they all relate to the same fungus. On woody plants in Uganda it is described as forming black crusts and plates on and in the bark, and wavy black lines in the wood and bark of affected roots. Unfortunately, in both East Africa and Ceylon most of the plants on which *M. phaseoli* has been found, including tea, are liable to infection by various other fungi, which often coexist with the sclerotial fungus. So common, indeed, is this association that Dr. Small holds the *Rhizoctonia* to be the primary parasite in all these root diseases of woody plants, the others being secondary followers that at most hasten the destruction of the roots. That the mycelium of some of these has been at times confused with the growth of *M. phaseoli* is clear from the mention both by Shaw and Small of clamp-connections in the hyphæ, pycnidial fungi being devoid of such organs, and it is possible that the crusts and black lines are equally due to mixed infection. Black lines are common in tea roots in Nyasaland on which sclerotia are found, but the latter are so frequently present without any trace of black-line formation that I doubt the connection between the two. In an attempt to distinguish between the black lines formed by *Armillaria mellea* and *Ustulina zonata*, specimens of the former on beech wood from Scotland (where the latter is unknown) and of the latter on rubber from Malaya (where *Armillaria* has not been reported) were compared and were found to be identical. This indicates the impossibility of determining from the characters of the lines in the wood the organism by which they were formed, and it must, for the present, be regarded as doubtful whether *Rhizoctonia* is capable of causing black lines in tea roots.

Besides the typical minute sclerotia, pure cultures isolated from which agreed with those of *M. phaseoli* from various sources grown at the Imperial Bureau of Mycology, other more irregular, often more prominent and shiny, and usually somewhat larger sclerotioid bodies were not uncommonly found on the dead roots and collars of bushes of all ages. Some of these appeared to be associated with pycnidial fungi other than *Macrophomina*. As cultures from these small stromata were not obtained, it was not possible to determine their identity.

Fungi of the genus *Rhizoctonia* are well-known as amongst the causes of damping-off in seed-beds, and the tea seedling disease above mentioned may be included in this category. They are favoured by overcrowding, insufficient ventilation, and excessive moisture. The tea seedling disease associated with *Rhizoctonia bataticola* is, so far as my observations went, usually not severe enough to justify special measures for its control. Where it is found attacking a considerable number of the seedlings, watering should be reduced and if the bed is shaded the coverings should be raised so as to allow the air free access to the beds. It might be advisable to remove them completely at night. The beds should not be sited in localities where there is not a reasonably free circulation of air, and tall grass or the like should be cut down in their immediate vicinity.

Seedlings that have died back in the manner described should not be used for planting out, even when they subsequently put out new shoots, as it is highly probable that the presence of the fungus on older plants is in part due to mycelium and sclerotia transferred with the plants from the seed-bed.

Attacks on older bushes cannot be prevented by any practicable measures, but they appear to be so sporadic as to be of little importance. It is a safe rule to remove all dead bushes when noticed in order to prevent the accumulation of root fungi in the tissues and their subsequent dissemination into the soil.

One other *Rhizoctonia* infection of considerable interest was found on a seedling that had died a year or two after planting out. The roots were covered with a violet growth in long twining strands or thin sheets. This was the mycelium of a fungus agreeing in all essentials with the well-known *Rhizoctonia crocorum*, the cause of the violet root rot of many plants in temperate countries and occasionally in the tropics. However, as *R. crocorum* has recently been shown to be the sterile stage of *Helicobasidium purpureum*, whereas an allied but distinct species, *H. longisporum*, is known on the roots of cacao and rubber in Uganda, it is possible that this *Rhizoctonia* belongs to the latter fungus. There was little doubt that the bush had been killed by its attack, but only one case of the kind was seen and the disease appears to be of no importance at present.

Fig. 7.—An obscure tea disease. Bush attacked from one side.

Fig. 8.—An obscure tea disease. Bush in late stage of attack.

Fig. 9.—Tea canker associated with *Macrophoma theæ*.

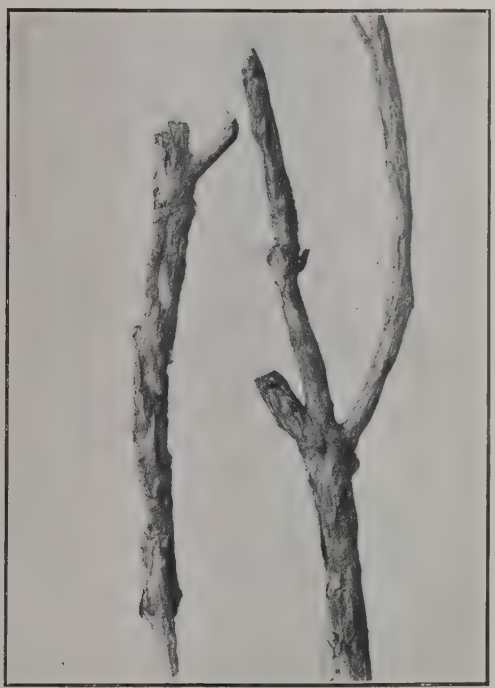

Fig. 10.—Tea canker associated with *Macrophoma theæ*. Late stage.

An Obscure Tea Disease.

Reports received indicated that the most serious root disease of tea in Nyasaland was that attributed to *Botryodiplodia theobromæ*, and it was soon evident that a large proportion of the patches of unhealthy or dead tea in the estates visited had many of the characteristics of internal root disease.

These patches were very irregular in shape, and whether they started from a single or several foci of infection, they nearly always showed several areas near together in which all or most of the bushes had died, while between these many of the bushes were healthly. The bare areas were connected with one another by alleys or lines of bushes which were dead or unhealthy, and beyond the limits of the patches single cases, apparently representing advance infections which often missed the nearer bushes to attack those further away, were found. The attack may commence in any part of a garden, except in the clay hollows ("dambos") previously mentioned, and though, in time, a great part of the garden may appear sickly, the advance is always roughly centrifugal and the parts furthest from the original attack are the last to become affected.

This disease appears to be responsible for more injury than all those previously mentioned taken together. It was the only disease causing spreading patches seen in the Cholo area and was the cause of exceedingly grave losses in some Mlanje estates. When of several years' standing, the total number of bushes affected in each attack ran into hundreds, and supplying with healthy seedlings had not checked the losses since the new plants were often attacked in their turn.

The above-ground symptoms are somewhat similar to those described in the internal root rot of tea first seen by me in Assam in 1902 and of which a short account was published in Watt and Mann's "Pests and Blights of the Tea Plant," 2nd Ed., pp. 414-416, 1903. The first sign is the development of shoots bearing small, narrow, pale green leaves, often with their edges upturned. The pale colour is due to a slight yellowing of the intervenous parts of the leaf blade, the veins remaining a darker green colour so that they show up prominently, just as in the Assam disease. As these shoots are not plucked, the bush soon assumes a twiggy appearance. In spreading patches these symptoms may develop on one side only (fig. 7), usually that from which the patch is advancing, but the side attacked does not die quickly as in unilateral infection by *Armillaria mellea* or *Ustulina zonata*, being marked, during the first rainy season at least, only by the pale narrow leaves and thin twiggy growth of the affected parts. In the following dry season these twigs may die back, but in the next rains a new growth of thin branches with few and still smaller leaves may partly replace them, while the rest of the bush develops the early symptoms of attack. In the second dry season most of the branches may die, but the rains again lead to the development of a few leafy shoots (fig. 8). The third dry season may see the bush killed, though it appears probable that several years often elapse before death takes place. This course is much slower than that of the other diseases described above except internal root disease, though the spread of infection from bush to bush is at least as quick, with the result that a considerable area may appear yellow and sickly even when the patches of dead bushes are of small size.

In certain areas the disease was said to be associated with the previous presence of particular trees, the commonest being *Parinarium mobola*. In one case seen nearly all the bare patches where bushes had been killed were situated so as each to encircle one of some half a dozen isolated, large, living *Albizzia fastigiata* trees, but the bushes between these were mostly in various stages of the disease and many were dead or dying.

In the early stage of attack the roots appear to be free from any fungous parasite. A complete examination of the root system from the collar to the finer feeding roots was carried out in several typical cases in the first year of the disease and nothing whatever was found that would explain their condition. The bark appears smooth and it and the wood are full of sap. They separate from one another unusually readily, appear to be more succulent, and the wood cuts more easily than in normal plants. The cells of the inner cortex and outer layers of the wood of the medium and smaller roots are often filled with a gummy substance together with flakes of a harder material. In the fleshy feeding roots the usual internal mycorrhiza of tea (the type with vesicles and arbuscules) is richly developed and the inner tissues are obviously healthy and functioning normally, but the surface of these roots and of those next in order may be irregular and roughened as if sucked or irritated by nematodes or something of the kind. A few roots were found, as might be expected, to contain occasional hyphæ, but these were not always of the same type and were altogether too few to account for a serious disease. Numerous attempted isolations from the roots gave quite inconclusive results, yielding no one fungus consistently and in some cases nothing at all. *Botryodiplodia theobromæ* was only once found in a single root, in these early cases, and it is impossible to attribute the disease to its attacks, though Captain Smee informed me that it is

common on bushes that have died in the manner described (which is very like certain cases attributed to this fungus) during the hot dry period of the year. Bushes killed by, or in the last stages of this disease that I examined showed various fungi in their roots including *Botryodiplodia*, but their general absence in the earlier stages makes it impossible to regard them as its cause.

The disease is locally regarded as a root disease and it is difficult to avoid this conclusion in view of the obviously spreading nature of the affected areas and their reported association with the previous presence of certain forest trees. Except for its unusually irregular spread, it has all the characters of a progressive underground infection by a parasitic fungus, though none such was found. The only other disease that I am acquainted with that spreads in a somewhat similar way and that apparently is not a root disease is the spike disease of sandal (*Santalum album*) in southern India, and this belongs to the most obscure group of the so called virus diseases, a group of which peach yellows in the United States is the best known example. Though the upcurling of the leaf margins in affected tea bushes and their mottling with pale and darker green suggests a virus disease, I am not aware of any that spreads so gradually and continuously; the leaf mottling is only due to retention of colour by the veins longer than the rest of the leaf, being thus unlike that found in the mosaic diseases; and there is no accumulation of starch as in peach yellows and spike disease of sandal.

Virus diseases, with a few exceptions, are ordinarily transmitted from plant to plant by insects. The exceptions include tobacco mosaic, where human agency is probably responsible for most of the spread, and, perhaps peach yellows and sandal spike, where the method of transmission is unknown. In a few instances, the chief of which is raspberry mosaic, the insect vector may be so sluggish that spread is continuous from plant to plant mainly by passive dissemination of the insect (as by means of the wind or by persons passing through the bushes and conveying green fly from one to another on their clothing). Some such method of spread must be responsible if the tea disease here described is due to an insect-borne virus.

In the absence of knowledge of the cause of this disease it is difficult to suggest methods of control. Observations indicate that it is worst in gardens where the soil has been exposed to wash or where the bushes are enfeebled from any other cause. The general measures for the indirect control of root diseases by improving the vigour of the bushes discussed below should, therefore, have an effect in reducing the losses caused by this disease. In advanced cases it is hopeless to attempt to save the bushes, and they should be removed and the patches treated as recommended for *Armillaria mellea* and *Ustulina zonata*. Whether earlier removal (as soon as the first symptoms are observed) should be recommended is doubtful on present knowledge. Experiments should be carried out in new extensions to test the effect of thorough rogning (eradication of every diseased bush at an early stage) on the spread of the disease, but such experiments should be under expert supervision if they are to yield reliable results. Stumping has been advised on account of the other diseases, and the reported association of this disease with certain tree stumps may indicate that this measure may be useful also against it.

There are many instances of successful control of a plant disease, the cause of which is unknown. Most of them are based on observations regarding the incidence and spread of the disease. Such information is very scanty in the present instance and requires to be supplemented by careful observation, pending an opportunity for more detailed research into the cause of trouble. It is not unlikely that the disease occurs in the East but has been confused with the effects of *Botryodiplodia theobromae*, and it is hoped that the above description may call the attention of pathologists in other tea growing countries to what is unquestionably one of the most serious diseases to which the tea bush is liable.

General Recommendations for Improving Root Development.

These should be directed primarily to checking soil wash, improving drainage, and maintaining soil fertility or restoring it when depleted.

The soils of the Mlanje tea area lend themselves particularly well to the checking of wash by the use of blind contour drains, the soil from which is thrown up along the upper face of the drain to make a low ridge which catches much of the silt and in time helps to form level terraces. Catch drains of this type were seen easily to hold the water in a fall of nearly nine inches in 24 hours, percolation in the open Mlanje soils being sufficient to prevent the drains from filling. In this case the slope was relatively gentle and the vertical distance between the drains was about 3½ feet. The various types of silt pits and terraces that may be used to check erosion are fully discussed by Captain Hornby in the Bulletin already referred to, and the details of their construction are outside my scope.

Cover cropping is the second main method of checking wash, and the extended use of cover crops in the Mlanje tea gardens, especially where there is little shade, would probably have a beneficial result. Little experimenting has been done in Nyasaland with cover crops suitable for tea, but many have been tested in Ceylon,[1] and some of those that have proved satisfactory there, especially species like *Indigofera endecaphylla*, belonging to genera common in the country, would probably be equally so under Nyasaland conditions. The period of the year when the cover is most required is December, as the first heavy rains, while the soil is bare after the hot weather and before the weeds have grown, do the greatest damage.

Green manuring and deeper cultivation have a two-fold effect in that they not only improve fertility but increase the water absorbing and holding capacity of the soil and thus check wash due to the run off of the surface water. A good many green manures have been tested,[2] but there appears still to be room for the introduction of low-growing leguminous crops that will keep down weeds, cover the soil well, and not impede the growth of the bushes. The "mposa" bean (*Phaseolus mungo*) was the most nearly satisfactory in this respect of those seen actually in use in the tea. In most gardens, however, green manuring is not practised, and its extended employment is probably one of the best methods of improving the surface development of the roots.[3]

Drainage is well known to improve root development and to lessen susceptibility to disease. The extended range of the root system in well drained soils helps the plant to resist drought conditions and to survive the loss of the surface roots from whatever cause, unless the taproot or collar of the bush becomes involved. Drainage and cultivation are the two main factors that render soil unsuitable for the growth of such forest fungi as *Armillaria mellea* and *Ustulina zonata*, and their neglect is probably the chief reason for the persistence of these parasites in gardens that have been cleared from forest for many years.

Fertilizers have been little used in Mlanje tea, under the mistaken belief that the soils are sufficiently rich to make them unnecessary. Whatever chemical analyses may show, there is sufficient evidence that the surface soils are, in general, in need of improvement, and in a great many gardens the use of fertilizers is clearly indicated. Phosphatic fertilizers combined with green manuring have a well-known effect in increasing surface root development and thus improving the health of the bushes. The Nyasaland Department of Agriculture is in a position to advise on the most suitable fertilizers for the different types of soil. In some of the gardens that have suffered most severely from soil wash there is no doubt that the restoration of fertility will be an exceedingly difficult task and will require to be carried out on the lines adopted for the renovation of old, worn-out tea soils in the East.

These matters have been touched on only in their bearing on the incidence of root diseases in the tea. Attention to them is at least as important as any direct methods of combating these diseases, such as stumping, trenching, and the removal of dead bushes. All the diseases encountered are present throughout the district, and the difference in their severity depends in great part on the manner in which the vigour of the bushes has been maintained. Neighbouring gardens may be seen, in one of which the losses from disease are relatively slight because the health of the bushes has been maintained by checking wash, fertilizing, and good soil and bush management, while in the other the condition of the tea has become so serious that it is doubtful whether it can be renovated without an almost prohibitive expenditure. There is probably no estate in the district which would not become as bad as the worst now visible in respect of root diseases if neglected, but, on the other hand, there are few that cannot be brought back into good condition by methods directed to remove the causes of ill-health discussed above.

Stem and Branch Canker (? Macrophoma theæ Speschnew).

The stem canker or die back associated with the presence of the fungus *Macrophoma theæ is* the only disease of consequence found on the above-ground parts of the bush in Nyasaland. It was first observed in February or March, 1926, when it caused much damage by killing back the shoots of the older bushes and leading to the death or serious injury of young plants and nursery seedlings. The attack was worst in hollows where the soil is moist and badly drained, and it was reported by Mr. E. W. Davy, Assistant Director of Agriculture (from whom the first specimens of the disease were received at the Imperial Bureau of Mycology), that there was no doubt that plants

1 *Cf.* Holland, T. H., in *Tropical Agriculturist*, LXVI, p. 248, 1926; LXVIII, p. 263, 1927.

2 Davy, E. W., "Crops for green manuring and soil protection."—Nyasaland Agric. Dept. Bull. 3, 1925.

3 *Cf.* Carpenter, P. H., "Green crops."—Quart. Journ. Indian Tea Assoc., 1926, part 4, pp. 170-175, 1926. The extensive literature on green manuring of arable crops in India is summarized by A. W. R. Joachim in *Tropical Agriculturist*, LXV, pp. 325-331, 1925.

raised from imported Indian seed were far less resistant to the stem canker than those from acclimatized Mlanje seed. Mr. Davy reported that Captain Smee had found a *Macrophoma* very commonly on the cankered areas, and the latter stated subsequently that this fungus was also found on *Grevillea* trees in the affected areas.

Isolations made from Mr. Davy's specimens yielded chiefly a species of *Macrophoma*, differing from *M. theicola* Petch, the cause of a stem canker in Ceylon, but agreeing in measurements with *M. theæ*, which Speschnew described as the agent of a leaf spot of tea in the Caucasus.[1] A species of *Tuberculariа*, resembling that belonging to the *Nectria* that causes die back of tea in northern India, was also present on some of the branches. Cultures of both these fungi were taken to Nyasaland in order to attempt to produce the disease by inoculation.

During my visit this disease was little in evidence, no doubt as a result of the drastic treatment carried out during the previous year. One affected garden of young tea, however, had not been heavily pruned to eradicate the cankers, and in this a number of cases were seen. The affected bushes each had one or more withering branches with yellowed or falling leaves. When these branches were more closely examined they were found to show swollen cankerous areas (fig. 9) involving sometimes a considerable length of stem, sometimes only the neighbourhood of the lateral branches or the bases of the latter. The thickening surrounds the entire stem, as a rule, and its surface is marked by a longitudinal cracking of the bark, which is often detached in flakes. Through the cracks there may be a protrusion of reddish woody tissue, which forms irregular cushions so that the swelling is not of uniform thickness all round the stem. At the time of my visit (February-March) true cankers exposing the original wood were rare, the infections having evidently not progressed very far, but in the material collected by Mr. Davy the previous July typical cankers were not uncommon (fig. 10). When the shoot is entirely ringed and the bark is destroyed down to the wood, the branch dies back above the point of attack, and this die back of the shoots is the main symptom later in the year. The cankers are less regular in appearance than those caused in Ceylon by *M. theicola*, the callus ring surrounding them is less uniform and less prominent, and they are often partially covered by shreds of bark. They are, however, exceedingly like one of the forms of branch canker known on tea in India[2] without any clue having, as yet, been found as to its cause. Infection appears to take place just below the insertion of the lateral branches (possibly through the leaf scar) and to extend usually both up and down the main stem and also out along the lateral branch. The longer cankered areas are usually caused by the union of several distinct infections. The surface of the cankered area may show the small black pycnidia of *M. theæ* or less often, the light pink cushions of the *Tuberculariа*. Other fungi are also found, but only one of these belongs to a genus (*Pestalozzia*) known to cause tumours or cankers, and as the species agreed with the common tea parasite *P. theæ*, which is not capable, so far as is known, of producing similar effects, it was not further considered.

The structure of the thickened stem tissues is of considerable interest. The greater part of the increased diameter is due to the formation of gall wood on the inside of the cambium. A much smaller increase takes place in the layers external to the latter, phloem and cortex. At a certain moment in the growth in thickness of the shoot, the cambium is stimulated to an excessive activity and commences to form on its inner face cells differing in their subsequent development from the normal elements of the wood. The new tissue thus formed is, therefore, heteroplastic, in Küster's sense of the word.[3] There is an increased production of parenchyma, the medullary rays are broader and less sharply defined, and the wood fibres and vessels may be few at first, as compared with the normal wood. The result is a kataplasmatic gall,[4] the bulk of the new tissue being gall wood. At the same time, there is a slight increase in the tissues external to the cambium ; these may become twice as thick as the normal bark, but the growth pressure exercised by the expanding gall wood causes splitting of the outer bark and the formation of irregular cork layers deeper in, so that finally there may be little cortex left. The expanding medullary rays may sometimes be traced across the cambium into the phloem, and in the latter, as well as in the deeper part of the cortex, stone cells and cells containing large crystals, almost entirely filling them, are developed.

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1 Speschnew, N. N., "Die Pilzparasiten des Teestrauches."—Berlin, Friedländer, 1907, p. 17.

2 Butler, E. J., "Fungi and disease in plants."—Calcutta, 1918, p. 463, and fig. 24 on p. 83. The latter figure would pass equally well for the Nyasaland canker in a rather early stage.

3 Küster, E., "Pathologische Pflanzenanatomie," 3rd Ed., p. 344, 1925.

4 *Ibid.*, p. 187.

Sometimes the cambium is killed and the overlying cortex dies, the resulting wound being covered more or less completely by an overlapping growth of the gall wood and of tissues exterior to the latter. If only a small part of the cambium dies, and the surrounding tissues are growing rapidly, a fissure entirely closed in may be formed in a part of the ring between the normal and the gall wood. In severer injuries, and especially at a later period of the year, the wound remains as a gaping canker with swollen callus lips around it. No alteration ever seems to take place in the original woody cylinder until open cankers lead to its decay, and the pith of the stem is also unaltered in appearance.

As a result of the irregular growth of the gall wood, especially where fissures are produced, its elements do not pursue a regular radial course. The medullary rays and other radially disposed elements in the normal wood become laterally displaced or curved, and may even eventually lie transversely to the radial axis. A curious feature observed in some of the galls is that while the cells nearer the sound wood are mostly parenchymatous and relatively little differentiated though with strongly pitted or reticulated walls, those further out, near the cambium (and, therefore, the most recently formed), resemble normal wood with clearly differentiated fibres and vessels In such cases it would appear that the stimulus that causes the development of the gall wood acts only for a time.

Colourless hyphæ occur in a few of the outer layers of the gall wood and in the phloem, in other words immediately on both sides of the cambium. In young galls, sections taken near the limit of the thickened part may show no hyphæ, even though the ring of gall wood is already clearly defined. In some galls no hyphæ were seen until nearly a quarter of an inch behind the part of the stem in which the new wood could first be distinguished. This suggests that the stimulus leading to the formation of gall wood acts beyond the limit of the area occupied by the fungus. The hyphæ found at the advancing edge of the infection were large "exploratory" hypæ, running longitudinally in the outermost vessels. Further back, more numerous, smaller hyphæ occur in the outer vessels, the neighbouring parenchyma, and the phloem tissues. They are always hyaline, intracellular, predominantly longitudinal in direction (except in the medullary rays), and sparingly branched. Sometimes even in fairly early stages, similar hyphæ can be found in the outermost vessels of the normal wood internal to the gall wood. At this period, however, hyphæ were not found in any other part, the pith, the cortex, and all the normal and gall wood except the regions above specified being quite free from infection. In older material, however, the mycelium spreads throughout all the tissues and is readily found from the cortex inwards to the pith.

There is little doubt that this mycelium is the cause of the disease. Its distribution, corresponding so exactly with the tissue stimulated to abnormal activity, and its constant presence a short distance behind the advancing limit of the stem thickening, tell strongly in favour of a causal connection. It is less certain that this mycelium is that of *Macrophoma theæ*. It has not been found possible to trace a direct union between the internal mycelium and the external pycnidia, and the single series of inoculation experiments undertaken with pure cultures failed, as did the similar ones made with the *Tubercularia*. A single failure, of course, is not conclusive, as there is no exact knowledge of the part of the stem through which entry is normally effected (leaf scar and wound inoculations on young branches were tried) nor of the season of the year at which the disease starts (the inoculations were made on 24th March, by which time the disease had already been in evidence for some months). It appears probable, on the whole, that *M. theæ* is the cause of the disease, not only because it was the fungus most commonly isolated from the cankers but also because *M. theicola* is already known to cause a somewhat similar disease while *M. tumefaciens* [1] forms tumours which are highly suggestive of a stimulatory action like that described above. In culture the mycelium of *M. theæ* would pass sufficiently for that present in the galls, except that with age the hyphæ become brown and brittle. In old cultures, too, the spores eventually become brown and septate, resembling those of the genus *Hendersonia*. Speschnew's fungus was found only on the leaves, and there are several cases in which species identified as *Macrophoma* have subsequently been found to develop septate spores when old, so that this fact by itself does not invalidate the identification of the Nyasaland fungus with the Caucasian one.

When the stem canker first appeared, Mr. Davy advised cutting the affected nursery seedlings back to three inches of stump, i.e. well below the cankered area, followed by heavy spraying with Bordeaux mixture and a subsequent lighter spraying when new growth commenced. On the young, recently planted out tea he recommended hard pruning, with spraying, and thorough draining of

1 Hubert, E. E., "A new Macrophoma on galls of *Populus trichocarpa*."—*Phytopath.*, V, pp. 182-185, 1915. Hyde, K. C., "Anatomy of a gall on *Populus trichocarpa*."—*Bot. Gaz.*, LXXIV, pp. 186-196, 1922.

the affected areas. In older gardens pruning out diseased branches was considered to be sufficient, accompanied by manuring. Further recommendations from the Bureau of Mycology included measures intended to promote improved root development and the application of potash. In affected nurseries attention has been drawn by Captain Smee to the necessity of improving soil conditions and, especially, the ventilation of the seedlings by opening up the vegetation surrounding the nursery site.

As already stated these measures, which were vigorously applied, appear to have been completely effective. In the absence of more exact knowledge of the life-history of the organism responsible for the disease they appear to be all that can be recommended.

2. Diseases of Tobacco in Nyasaland.

The climate and soils of a great part of Nyasaland are suitable for the growth of tobacco, the area of which under European management amounted to some 25,000 acres in 1927 with, perhaps, half as much in addition grown by the natives. The quality of the leaf is, in general, good and Nyasaland tobacco has found favour with the trade, so that, aided by imperial preference, there was a ready demand for the bumper crop that was being harvested during my visit. Both bright and dark tobacco are produced, amongst the varieties cultivated being the Rhodesian Hickory Pryor, Gold Leaf, White Burley, Melton, Western, etc.

Only a few of the tobacco plantations in the Shiré Highlands were visited owing to lack of time, so that it was not possible to attempt a survey of the most prevalent diseases, and the following notes are necessarily incomplete. It appears probable, however, that most or all of the tobacco diseases recorded in Southern Rhodesia and the Union of South Africa also occur in Nyasaland and that disease control will not differ materially in this large tobacco belt, allowance being made for seasonal differences in rainfall and the like. The first disease described below, however, has not been reported elsewhere in southern or eastern Africa, and its control will require to be worked out locally.

Black Stem Rot due to Pythium aphanidermatum.

A disease of tobacco seedlings was observed at Pusa, India, about 1916 and was found to be due to a species of *Pythium* to which the name *P. butleri* was given.[1] The same fungus caused a rot of ginger (*Zingiber officinalis*) rhizomes, which had been earlier attributed to *P. gracile*, and of the base of the stem of papaw (*Carica papaya*), cross-inoculations showing that it was able readily to pass from one host to another. Subsequently, strains of what appears to be the same organism were found attacking several cultivated cucurbits at Pusa.[2]

Some time earlier, in 1915, a similar fungus was described in the United States as a parasite of radishes and sugar beets, under the name *Rheosporangium aphanidermatus*.[3] Later on, a root disease of sugar-cane caused by an organism which was identified as *P. butleri* was studied in Hawaii, and the suggestion was made that *R. aphanidermatus* was the same species.[4] This suggestion has been accepted by recent American workers and the name has been changed to *Pythium aphanidermatum*.[5] Diseases of various cucurbits, peas, and eggplants have since been found to be caused by it in America.

In 1922 a black stem rot or stem scorch of tobacco plants after transplanting was reported from Deli, Sumatra, where it caused so serious an outbreak in certain fields that scarcely a plant escaped.[6] Several species of *Pythium* were said to be able to cause it, one of these being at first named *P. butleri* and subsequently *P. aphanidermatum*.[7]

Specimens of a *Pythium* said to cause a tobacco disease were sent to me from the Gold Coast in 1923, and were identified as *P. aphanidermatum*,[8] the question of the distinction of this fungus from *P. butleri* being left over for comparative study, which has not hitherto been possible.

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1 Subramaniam, L. S., "A Pythium disease of ginger, tobacco, and papaya."—*Mem. Dept. Agric. India*, Bot. Ser., X, 4, 1919.

2 Scientific Reports, Agricultural Research Institute, Pusa, 1924-5, pp. 52-53, 1925.

3 Elson, H. A., "*Rheosporangium aphanidermatus* a new . . . fungus parasitic on sugar beets and radishes."—Journ. Agric. Res., IV, pp. 279-291, 1915.

4 Carpenter, C. W., "Morphological studies of the Pythium-like fungi associated with root rot in Hawaii."—Bull. Exper. Stat. Hawaiian Sugar Planters' Assoc. Bot. Ser. III, pp. 59-65, 1921.

5 Fitzpatrick, H. M., "Generic concepts in the Pythiaceæ and Blastocladiaceæ."—*Mycologia*, XV, pp. 167-168, 1923.

6 Sidenius, E., "Verslag van het Deli Proefstation 1921-2."—Meded. Deli Proefstat. te Medan-Sumatra, Ser. 2, XXIV, p. 35, 1922.

7 Jochems, S. C., "Parasitaire stengelverbranding bij Deli-tabak." *ibid.*, XLIX, 1927.

Bunting, R. H., and Dade, H. A., "Gold Coast Plant Diseases."—London, Waterlow and Sons, pp. 87-88, 1924.

In Nyasaland a tobacco disease with the characters of the black stem rot of Sumatra was found by me in the Cholo, Mlanje, and Zomba districts. The fungus associated with it was indentified as *P. aphanidermatum*, and was quite similar to the published description of this species and also to the Gold Coast specimens, except in the oogonial and antheridial characters referred to below. In the most severe attack seen, the field had been cleared from forest the previous year, and the majority of the nearly mature plants that it contained at the time of my visit were wilting just as in a severe attack of root knot due to the nematode worm *Heterodera radicicola*. There was too little of the latter, however, to account for the diseased condition, and the owner had accordingly reported that his crop was attacked by an unfamiliar disease. In the Zomba district also the disease was recognized by planters as one not previously described in Nyasaland when first observed during the current year. Subsequently it was found in a couple of Mlanje plantations, and it may be presumed that it is present to some extent throughout the Shiré Highlands.

The disease causes a flaccid drooping of the leaves of the whole plant, most noticeable late in the day, the plants recovering to a certain extent during the night in the early stages. This wilting is unaccompanied at first by any browning of the leaves or upper part of the stem. As a rule, it is not very evident until the plants are nearly mature, but after topping, the symptoms rapidly develop. In the flowering stage, the flower stalk is the first to droop and soon falls over. In one affected area the plants were said to have never looked well after planting out, but this was attributed to the use of over-mature seedlings. In another, the leaf scars left after removing the basal leaves when transplanting showed a brown patch running down the stem; about three weeks after transplanting, a period of dry weather caused a check to the plants and though they came away again a certain number never grew normally and wilted when approaching maturity. In this instance also the trouble was traced back to the seed-beds, since three separate areas planted from the same nursery all developed the disease, others from different nurseries being less affected.

All the affected plants were marked by sunken black or dark brown areas at the base of the stem and extending for some inches above the soil level (fig. 11), several such patches sometimes being found on a single plant. The interior of the cortex ultimately becomes jet black under the sunken patches, which are due to a dry rot that causes the collapse of the cortical cells without, as a rule, any rupture of the epidermal layer. On splitting the stem the pith is also frequently blackened and usually is hollow and divided into chambers by transverse discs of tissue, the cavities being occupied by a loose growth of white, cottony mycelium. A similar mycelium may be found around the base of the stem, or can be developed by keeping the plants moist after pulling them up. The woody cylinder of the stem remains clean and white until it becomes invaded by secondary organisms. In a few cases infection was found to have spread from the stem into basal suckers, the leafy buds of which were blackened and rotted by the fungus.

A hyaline, unseptate, profusely branched mycelium occupies the cortex, the main hyphæ running between the cells and sending lateral branches into them. These intracellular branches tend to remain confined to the cells, which are often filled with coiled hyphæ, apparently unable to pass through the cell walls (fig. 12*c*). The wood is usually free from the fungus until a late stage in the disease. In the loose mycelial wefts found in the cavities of the pith or when an infected stem is incubated (occasionally also in the cortical mycelium) swollen, lobed or shortly branched hyphæ are common, or a normal hypha may bear lateral swollen buds or diverticula to a greater or less extent (fig. 12*a*). When placed in fresh water septa develop, cutting off a variable extent of hyphæ bearing these diverticula, the part thus cut off becoming a sporangium (fig. 12*b*). The sporangia are indistinguishable from ordinary hyphæ except that diverticula appear nearly always to be present where they form. They mature rapidly and discharge their undivided protoplasm, through an opening situated usually at the tip of one of the branches of a diverticulum, into a very fine bladder or vesicle, in which the zoospores are fashioned. As the latter develop they commence movement, at first in a mass and then individually, the vesicle soon rupturing to allow the separate zoospores to swim away. The earlier formed sporangia are small and contain up to fifty rather large zoospores of the type normal in the genus *Pythium*, but later on much larger ones, containing some hundreds of spores, may form. In these, especially, the contents of the hyphæ furthest away from the mouth may divide into masses representing individual zoospores before they reach the vesicle or even before they begin to move towards it, thus resembling the normal process of zoospore-formation in the genus *Aphanomyces*. Edson[1] had already noticed that cleavage into zoospores may occur before the contents of the sporangium escape into the vesicle.

1 Edson, H. A., *loc. cit.*, p. 285.

Fig. 11. Black stem rot of tobacco caused by *Pythium aphanidermatum*, showing sunken black patch at base of stem and drooping of the leaves.
(From a painting by Mrs. A. E. Shinn.)

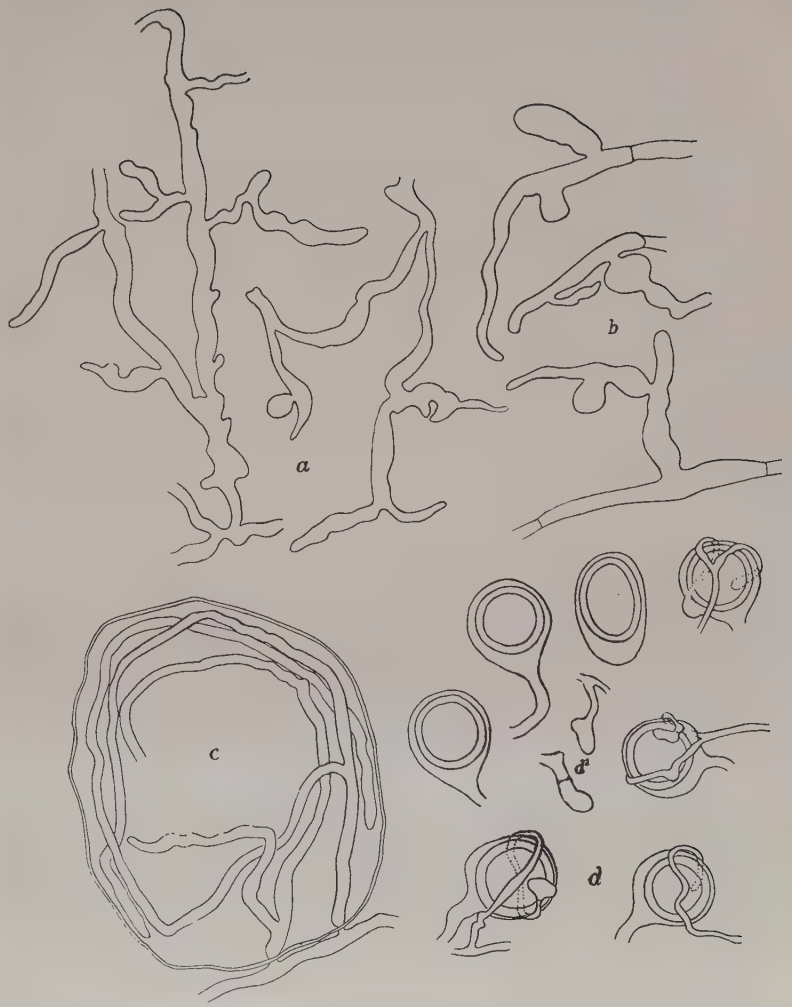

Fig. 12.—*Pythium aphanidermatum* from tobacco: *a*, Swollen mycelium from pith; *b*, sporangia; *c*, a pith cell with intracellular hyphae; *d*, oogonia, oospores and antheridia.

Oospores were found in the pith and surface mycelium on several occasions (fig. 12*d*). The oogonium is borne terminally on a hyphal branch and is irregularly club-shaped, measuring about 18 to 28 microns in diameter. It is fertilized by one or more antheridia borne on slender, coiled, often branched stalks which, in some cases at least, arise from a different hypha from that on which the oogonium is formed. The antheridial branches encircle the oogonium, usually nearly completely, before forming the terminal, rather small and irregularly shaped antheridium (fig. 12*d*1). The oospores lie loosely within the oogonium and are usually quite spherical, though sometimes elongated to fit the shape of the host cells. They are colourless, average 18 microns in diameter, (extremes 14.5 to 24 microns), and their wall is usually 3 to 3.5 microns thick. In its oogonial and especially, antheridial characters the fungus agrees more closely with that described by Carpenter on sugar-cane in Hawaii than with the original descriptions of *P. aphanidermatum* and *P. butleri*, but until more is known of this group of parasitic forms it seems unnecessary to give it a distinct name.

The description of tobacco stem scorch in Sumatra, the only other locality besides the Gold Coast and Nyasaland in which *P. aphanidermatum* has been reported causing a disease of tobacco after transplanting, corresponds fairly closely to that given above. In its most typical form it is said to appear soon after planting out, but infection may occur from this period up to a few days after full growth. Seedling infection has been occasionally seen and may involve the leaves as well as the stems. Infected young plants have their stems much shrunken near the ground level on account of a rot which may cause the plants to break or fall over. When more woody, the stem shrinkage is restricted to the cortex, and results in a sinking in of the stem in a broad patch, often confined to one side. In mild cases there may be only a few dark stripes on the stem. When cut, the cortical tissues of affected parts appear jet black. The hollowing and chambering of the pith are as described above, and a white, loose, woolly growth of mycelium is often found around the stem below ground level.

The worst attacks in Sumatra have been on land planted with *Leucaena glauca*, which is sometimes grown to protect the secondary jungle in fallow tobacco land from fire. Both this plant and *Phytolacca decandra*, also sometimes found in tobacco land, have been shown to be susceptible to infection and to transmit the disease to the tobacco crop. Infection is especially liable to occur when transplanting is done during wet weather; after dry weather during the period of transplanting little stem scorch appears. All types of soil are liable to infection, but the worst attacks appear to be in clay soils containing an admixture of sand. In the Gold Coast the disease occurs in hot, dry savannah land which is liable to waterlogging during the rains and becomes caked when dry. Tobacco growing on better soil in the neighbourhood escaped.

As the parasite belongs to the fungi that require films of free water for their dissemination, and as there is evidence that infection starts in the seed-beds, the chief preventive measures that can be recommended at present are to reduce watering and to allow the soil to dry somewhat immediately the first cases are seen in the nursery. At the same time the ventilation should be improved by raising the shade or removing it at night, and this will also help to dry the beds. Little can be done to check the disease after transplanting, except the removal of infected plants and their destruction by burning in order to prevent the carrying over of the disease to the following season. The oospores alone are capable of prolonged vitality, and as they are chiefly formed in the pith the destruction of affected plants should kill most of them. In Sumatra it is recommended that special seed-beds should be kept in areas subject to the disease, to provide supplies for replacing plants killed by it in the first few days after transplanting, when most of the attacks occur. These beds should not be watered for some days before their seedlings are required, in order to get more woody plants, and should be sown a few days earlier, so that the seedlings will be older by, say, five days than those normally used. Instead of having special seed-beds, it is sometimes enough to leave some seedlings behind when transplanting from the ordinary beds and to cease watering these for eight days or so before they are used. It is also advised to be careful, when planting out, not to bury the stem in the soil, since it is more susceptible to attack than the roots. The seedlings are placed in shallow holes with a little loose soil around the roots, and only watered sufficiently to secure growth, and then, eight or ten days later, the holes are filled and the soil drawn up around the plants. Most of the Sumatra attacks appear to be due to infection after transplanting, and they are so frequently associated with the previous presence of *Leucaena glauca* or, less often, *Phytolacca decandra* that the best remedy is said to be the eradication of these plants. The prevalence of *P. aphanidermatum* in Nyasaland is a strong argument against the introduction of *Leucaena glauca* as a green manure, but whether there are any other indigenous plants liable to attack by this fungus is not known.

Leaf Spots.

Tobacco suffers rather severely from various leaf-spotting diseases in Nyasaland. The commonest of these during the period of my visit was the so-called frog-eye spot, due to *Cercospora nicotianæ*. This is found chiefly on leaves that have become mature, and is thus ordinarily confined to the basal and middle parts of the plant. When the harvest is delayed a large proportion of the leaves may become infected. The bumper crop of 1927 was no doubt responsible for the many complaints of leaf injury from this fungus in the curing barns, as the labour force was not always sufficient to cope with the crop and a good deal of it was cut late. Under such conditions, even when the harvested leaves look fairly clean, a number of incipient infections are likely to have occurred before the leaves are cut, and these will continue to develop for some time in the barns. On several occasions a large proportion of the leaves in the barns was found to be badly spotted, the spots varying from the ordinary small, circumscribed ones, to large spreading patches which ultimately invaded a great part of the leaf. Unlike the spots formed in the field, most of which have a pale centre surrounded by a narrow brown line, the barn spots were uniformly dark in colour, with little or no zoning. Most of these spots remain without producing spores unless the leaf is taken out and incubated in a saturated atmosphere, when in two or three days a very copious growth of long conidiophores and conidia takes place. At first sight the fungus concerned is difficult to identify with *C. nicotianæ*, the conidiophores being very long (up to over 500 by 6 microns), unbranched, multiseptate, with few knee bends, and bearing very large conidia (up to over 300 by 4.5 microns at the base) at their apices. The knee bends are due to a further growth of the conidiophore from below the terminal conidium, the latter being pushed over to one side and soon falling off, while another terminal conidium develops at the apex of the branch. This long-spored form was first found in the Dutch East Indies and subsequently considered to be a distinct species, *C. raciborskii*. More recent workers in India, Ceylon, and the Dutch Indies, however, regard it as only a form of the American frog-eye organism, *C. nicotianæ*, and in these countries as well as in Rhodesia the latter is the name used.

A second barn spot of different appearance was seen in a few plantations, causing much damage. The spots were intensely black, often slightly raised and rough on the surface, small, and irregular in shape. At first they were taken for angular leaf spot (*Bacterium angulatum*), but in every case examined the superficial tissues were found to contain large quantities of dark, brittle hyphæ, often aggregated into stromatic masses, and responsible for the slightly raised surface of the spots. On incubation these spots gave the same *Cercospora* fructifications as those mentioned above. In some cases it was almost or quite impossible to detect these spots before the tobacco was put in the curing barns, though they were usually visible on holding the leaf up to the light as very fine points of lighter green than the rest of the tissues. It is possible that their subsequent development in a form so unlike that normally produced by *C. nicotianæ* is due to the special conditions of temperature and the like in the barns. Further work, however, is required before it can be concluded that all the spots that develop or spread in the barns are due to this fungus, though all those examined by me appeared to be associated with it. As a barn spot, under the conditions of the 1927 harvest, this disease is more destructive than in the field.

C. nicotianæ is a true parasite and, like most of the genus, not very easy to grow on dead materials, though able to retain its vitality for a considerable period on the withered leaves, after ceasing growth. Hence the method adopted locally to check its extension in the barns by raising the temperature to 120° F. or more, for a time, a method that is said to have given good results in many cases, probably acts rather by killing the leaf tissues than by injuring the fungus, which the temperature reached would be quite unlikely to do. If the leaf is killed too quickly, however, the curing process is apt to suffer, and great care has to be exercised or the remedy proves worse than the disease. Undoubtedly the chief way to avoid an excessive amount of spotting in the barns (a little is of no account) is to harvest the leaves before they are over-ripe. An additional precaution is to remove the affected basal leaves as they develop the spots and before the latter have time to produce spores which will infect leaves higher up. A Florida report states that frog-eye was very prevalent in the fields in 1922, but that where the leaves could be primed early little damage was caused. In Java, where the barn spot caused by this fungus has also been reported[1] rapid drying and improving the ventilation of the barns have given good results. It is probable that, as a field disease alone, this fungus is not ordinarily capable of causing enough injury to justify special measures of control, but it is a serious trouble in the curing barns, and the recommendations given above should be of value in preventing it from reaching the barns and checking its extension in the latter.

[1] Jensen, H., "Ziekten van de tabak in de Vorstenlanden."—Meded. Proefstat. Vorstenl. Tabak, XL, pp. 17-19, 1. 12, 1921.

Other tobacco leaf spots observed were associated with *Alternaria* sp. and *Phyllosticta nicotiana*, but they were of minor importance and usually associated with injury to the leaves during high winds or the like.

Both wildfire (*Bacteriam tabacum*) and angular leaf spot (*Bact. angulatum*) have been reported to occur in Nyasaland, and in a few cases spots were found that were probably due to one or other of these diseases. At the time of my visit, however, the tobacco was too far advanced for much evidence of the damage caused by them to be visible, and I was informed that neither had been prevalent in the current year's crop. Their characters and control are too well known to require description, but they are not always easy to distinguish from one another without careful microscopical and cultural examination. Work on these diseases is in progress in the neighbouring colony of Southern Rhodesia and the results will, doubtless, be applicable in the main under Nyasaland conditions.

Powdery mildew (*Oidium tabaci* or *Erysiphe cichoracearum*) was rather prevalent on some estates and is possibly gaining ground since the popularity of the Hickory Pryor variety has led to its extended cultivation. This variety is moderately susceptible and has replaced certain less susceptible varieties previously grown. As in other countries, the disease is worst in shady places and is associated with certain climatic conditions, including periods of hot dry weather. A careful watch should be kept on its spread, as it is scarcely possible to control it in any other way than by the growing of resistant varieties. Susceptibility to mildew cannot be judged by a single season's observation, but if infection is found to increase during a period of years, search should be made for resistant strains of Hickory Pryor or other suitable varieties to replace those now grown.

The disease is well known throughout southern and eastern Africa and in the East. In Java it has become of considerable importance, and serious efforts have been made to find a satisfactory method of control.[1] The only direct treatment that has given good results without injuring the curing properties of the tobacco is the application to the soil under the crop of sulphur at the rate of 150 lb. to the acre. This is far too costly to be practicable under Nyasaland conditions. In the early stages, the spread of infection may be checked to some extent by removing the first leaves that show the mildew. Wide spacing and free ventilation also help to reduce the severity of the attack.

Non-Parasitic Leaf Spots.

Various types of non-parasitic leaf spots are common on tobacco in Nyasaland, as in other countries. The most noticeable is the white speck, white rust, or pox disease, characterized by the development of large numbers of small, irregular, slightly sunken, white spots on the leaves. These may be confused with one form of the spotting caused by *Cercospora nicotianæ*, but in the latter the white spots are round and bounded by a narrow brown line, and they are seldom so small or so densely crowded as in the white speck disease. The latter also is found on leaves of all ages, instead of being, as a rule, confined to the middle and lower leaves of the plant.

Another type of spotting is that commonly known as red rust, of which there appear to be several forms. One of these, prevalent in Rhodesia and South Africa, is said to be caused by the fungus *Macrosporium longipes*, but most of the red rust seen in Nyasaland was not associated with any obvious parasite.

Both white speck and red rust are believed to be non-infectious and they usually affect only individual leaves or plants, not spreading from plant to plant. They, therefore, cause relatively little damage as a rule. Their cause is unknown and no methods of control have been described.

Virus Diseases.

Tobacco mosaic is at least as common in Nyasaland as in any other tobacco-growing country. Late in the season, after harvesting has been in progress for some time, it is usual to find practically every plant with mosaic symptoms in the lateral shoots and suckers that develop during this period. Fortunately, much of the infection is late in developing, and the yield and quality are not so seriously affected by the disease as might be expected. It is well known to all planters and it would appear that care is usually taken not to plant out mosaic infected seedling from the seed-bed. No method of controlling the disease in the field is known, and much work yet remains to be done on the means by which the disease is carried over from one season to the next in Nyasaland before any serious effort to check it can be made.

[1] D'Angremond. A., "Nadere gegevens over bestrijding van veldschimmel, etc."—Meded Proefstat. Vorstenl. Tabak, LVI, 1926 (English summary).

A second disease of the same group, which is relatively common in Nyasaland, is that variously known in other countries as ringspot, ringworm, or hieroglyphics. It was first described in Virginia but has also been found in other parts of North America,[1] in Sumatra,[2] and probably in South Africa.[3] That it is an infectious disease was conclusively established in 1927, when it was found to be readily transmissible from diseased to healthy plants by inoculation with the crushed leaf tissue, just as in tobacco mosaic. As no parasite has been found, it is regarded as one of the transmissible virus diseases, but with limited powers of dissemination so that natural spread from plant to plant is not marked.

Affected leaves are marked by white or pale rings or wavy lines, which often bear a definite relation to the veins, spreading out on each side of the latter in leaf-like patterns. The number of affected leaves on a plant and also the area of the leaf marked by the wavy lines vary greatly, but usually only a relatively small part of a few leaves is attacked. The effect on the leaf differs according to the variety of tobacco; in some there is nothing more than the often rather faint outline caused by the pale wavy lines, while in others the part of the leaf within the line turns brown and dries up. What is possibly only a form of the same disease is a brown decay of the veins on part of the leaf, which does not spread much into the surrounding tissue, but may lead to distortion and cause a good deal of injury at harvest time. On the whole, however, ringspot is not a serious disease, and it is only mentioned because it is one with which most planters are familiar so that what little is known about it is of interest.

The above notes cover all the tobacco diseases that were seen. None of them approaches in importance the root-knot disease caused by the worm *Heterodera radicicola*, which is by far the most serious tobacco trouble in Nyasaland. Some evidence was obtained that both the Granville wilt or slime disease due to *Bacterium solanacearum*, and the black root disease caused by *Thielavia basicola* occur in Nyasaland, but no serious attacks that could be attributed to them, or to the equally destructive *Fusarium* wilt of other countries, were seen.

1 Fromme, F. D., Wingard, S. A., and Priorde, C. N., "Ringspot of tobacco: an infectious disease of unknown cause."—*Phytopath.*, XVII, pp. 321-328, 1927.

2 Jochems, S. C. J., "Handleiding voor de herkenning en bestrijding van de ziekten van Deli-tabak."—Meded. Deli Proefstat. te Medan-Sumatra, 2nd Ser., XLIII, pp. 30-31, pl. 27, 1926.

3 Moore, E. S., "Diseases of Virginian tobacco in South Africa."—Journ. Dept. of Agric. S. Africa, XII, p. 454, fig. 20, 1926.

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MORPHOLOGY OF THE CHYTRIDIACEAN FUNGUS, CATENARIA ANGUILLULAE, IN LIVER-FLUKE EGGS.

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Morphology of the Chytridiacean Fungus, Catenaria anguillulae, in Liver-fluke Eggs.

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With nineteen Figures in the Text.

THE genus *Catenaria* was established by Sorokin in 1876 (9) for a Chytridiacean fungus which he found attacking eelworms, in a vessel of water with algae at Kazan, and named *C. anguillulae*. In this vessel, which was kept for several months, a large number of eelworms developed and were eventually killed off by an epidemic of fungous diseases due to several different Chytridiaceae and Ancylistaceae. *Catenaria* was described as resembling *Achlyogeton* and formed of a septate mycelium, segments of which swell up into sporangia, so that the thallus is transformed into a chain of sporangia separated by short lengths of hyphae. These isthmuses are generally bicellular. The sporangia open by a single beak or tube of discharge and liberate round zoospores, which emerge singly by creeping, and then, after a few seconds of immobility at the mouth of the sporangium, develop a single posterior cilium and swim off with a continuous rapid and circular motion. Sometimes two or three spores are liberated together or the whole emerge in a mass, but they quickly separate and commence individual movement. The empty sporangia are more or less oblong in shape (rounded and elliptical ones are also figured), and they have a distinctly double-contoured wall. They measured from 10 to 17 by 8 to 10 μ, the zoospores being 1·5 to 2 μ in diameter.

Not only was this fungus found in the bodies of the eelworms, but eggs of various aquatic 'animalculae' were also infected. Three of these, which are thought to be possibly rotifer eggs, are figured by Sorokin, two of them showing isthmuses between the sporangia, and the third a cluster of sporangia between which hyphae are not visible.

In 1885 and 1886 Dangeard (5, 6) recorded his observations on a fungus parasitic on eelworms that he considered to be the same as Sorokin's species, and also as that described by A. Braun in 1856 under the name *Chytridium zootocum* (1). Further, as Dangeard points out, it was clearly

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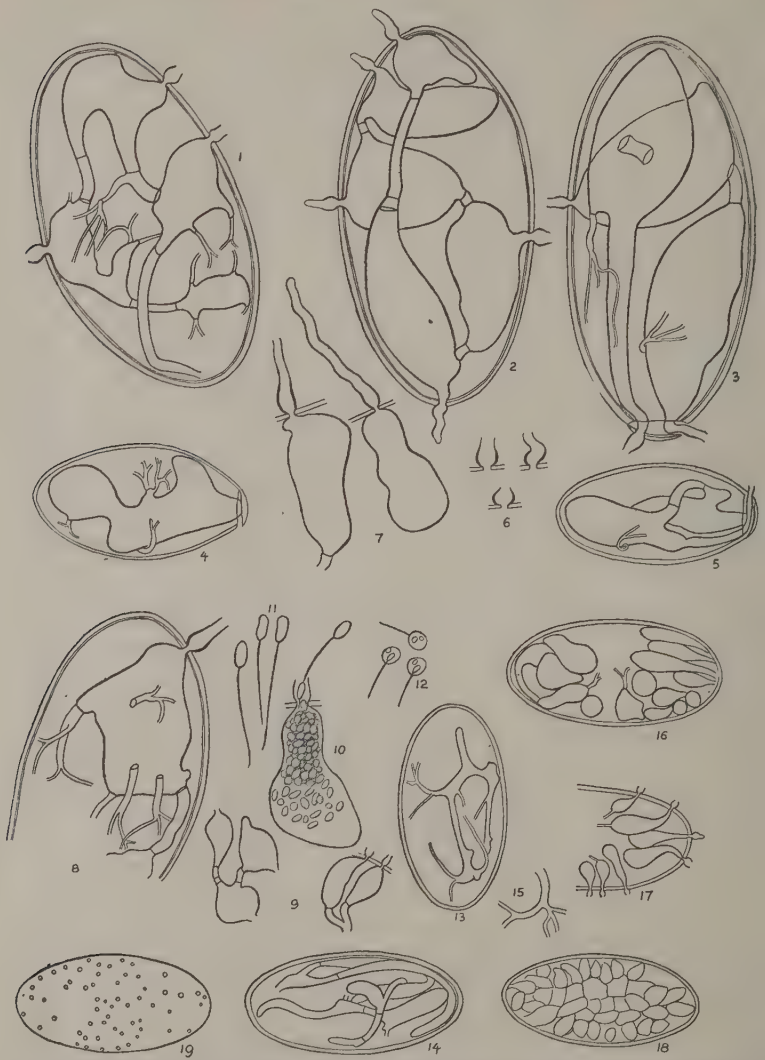
described and figured by A. Villot (10) some years before it was named by Sorokin. The organism which Villot observed and took to be a freshwater alga is, according to the latter writer, not rare near Grenoble as a parasite in several species of hairworms of the genus *Gordius*, and he gives references to the work of various earlier observers who have described similar parasites in this family. From an examination of his rather crude figures but excellent description, it is difficult to avoid the conclusion that the organism he studied was a *Catenaria*, and if so it would appear to be a common parasite of hairworms. Dangeard does not mention where his own material was obtained, but as he was at the time in the University of Caen, it was presumably from that neighbourhood. By placing some *Nitella tenuissima* in the vessels containing the infected eelworms, he obtained several thalli of the *Catenaria* in the algal cells, which it was possible to study with greater precision than those formed in the worms. In this way he was able to confirm Villot's observation that the thallus is dichotomously branched. He also established for the first time the existence of rhizoids, developed both from the isthmuses between the sporangia and from the latter themselves and forming a network of very fine, thin-walled filaments in the *Nitella* cell. These he had been unable to see in the bodies of the worms, though he had already strongly suspected their presence: he regards them as evidently nutrient hyphae, having the same function as the rhizoids of *Rhizidium* and *Cladochytrium*. Sorokin's statement that the mycelium is septate from an early stage is contradicted, the thallus being unicellular until the protoplasm becomes retracted to form sporangia. In the isthmuses the number of septa (as well as the length of the connecting hyphae) is not constant: there may be only one cross-wall or even none, in which case the neighbouring sporangia remain in communication. The little septum found in the rhizoids can only appear after these have ceased to function. The zoospores are liberated in two different fashions, either one by one with changes in shape due to the narrowness of the passage, or several together surrounded by mucus, which dissolves in a few seconds and sets them free. Sorokin's description of the manner of their exit is stated to be incorrect, since the cilium is already formed as they emerge, and may, indeed, often be seen before they leave the sporangium. The zoospores within the sporangia are shown to be more or less rounded, but after exit they are distinctly, though irregularly, elongated. Zoospores that are left behind may be found creeping around the walls in search of the opening by a sort of amoeboid movement, during which the spherical shape is lost. After ten minutes or so of active motion after leaving the sporangium the zoospore settles down, fixed on its cilium, and continues to wave in a jerky fashion for some time before coming to rest. Germination occurs by the emission at two opposite poles of the spore of one or several very fine filaments, which branch and form a mycelium with the spore, slightly enlarged

and with very watery protoplasm, in its centre. It may be noted that Villot's account of spore germination (loc. cit.) is in general agreement with this. Further enlargements take place in the filaments of the thallus and ultimately become sporangia. The latter are stated to be up to 32 by 20 μ in the large thallus figured in Pl. XIV of Dangeard's paper (6), but the single sporangium shown in Fig. 12 of the same plate can be calculated from the enlargement given to be 48 by 17 μ. In the same way the free swimming zoospore figured can be calculated to be 5·5 by 3·4 μ. The tube of discharge is stated to project little or not at all beyond the wall of the host as a rule; but it is sometimes of considerable length, as shown in Fig. 8 of his 1885 paper.

Dangeard called attention to the ease with which *C. anguillulae* may be mistaken, when overcrowding or lack of nutriment causes a reduced development of flask-shaped sporangia, for an *Olpidium*, and suggests that in some cases *O. endogenum*, A. Braun, might be incorrectly recorded if the rhizoids are overlooked. Similarly he questions whether *O. gregarium* (Nowakowski), Schroeter, may not be only a form of *C. anguillulae*, especially since both Sorokin and himself found rotifer eggs (the substratum of *O. gregarium*) attacked by a parasite resembling *O. gregarium*, but with mycelial elements or rhizoids, and with, in Dangeard's case (6, p. 287), zoospores measuring 4 μ in diameter and a long posterior cilium. As mentioned above, Sorokin identified this form with *C. anguillulae*, and Dangeard is inclined to agree while leaving open the question whether Nowakowski's fungus is not really the same. Later studies of *O. gregarium* (2) by the present writer indicate a somewhat wide divergence in the manner of emergence and shape of the zoospores from *C. anguillulae*, though rhizoids were not specially searched for and may possibly have been overlooked, the more so as great difficulty has been experienced in detecting them in *C. anguillulae*. Dangeard's suggested identification of the imperfectly described *Achlyogeton? rostratum*, which Sorokin found in the same batch of eelworms as *C. anguillulae*, seems also open to doubt in view of the entire absence of mycelial isthmuses between the sporangia.

In 1901 Constantineanu (4) observed in eelworms from the marshes near Jassy, Rumania, a *Catenaria* which he identified as *C. anguillulae*. He points out, however, that both the sporangia and their tubes of discharge are much larger than those described in Sorokin's species, the former being 32 to 54 by 21 to 29 μ, and the latter 54 to 243 μ long. His figure shows a chain of more or less oblong or triangular sporangia united by unicellular or bicellular isthmuses, and he states that the mycelium in the thalli observed was not branched. He appears to have been unacquainted with Dangeard's papers, where he would have found dimensions assigned to *C. anguillulae* approaching his own, and, in particular, tubes of discharge figured that are several times the length of the sporangium.



Catenaria anguillulae. FIG. 1. A thallus with four discharged sporangia and a copious formation of rhizoids mostly arising from the sporangia, in which case there is a septum cutting off the rhizoid, but in one case arising from an isthmus between two sporangia and then without a septum. × 430. 2. A thallus with five sporangia, one of which has just discharged, while the others are ripe. Rhizoids not shown. × 430. 3. A thallus with three large discharged sporangia, one of which has a very long neck. The swollen part of this sporangium was 50 by 36 μ, and the neck 60 by 9 μ. Rhizoids scanty. × 430. 4. An egg containing only a single large irregular sporangium with

These appear to be the only records of *Catenaria*, if Serbinov's *C. pygmaea* (8) be excluded. This species is regarded as doubtful by von Minden in the 'Kyptogamenflora der Mark Brandenburg' (vol. i, p. 419, 1911), and it is certainly difficult to include a form characterized by the development of only a single sporangium at the end or in the middle of a highly developed rhizoid system in a genus, the distinguishing feature of which is the formation of chains of sporangia separated by septate hyphal isthmuses. In this case there can be no question of dwarfing through overcrowding or lack of nutriment, as the fungus occupied the cells of *Mougeotia*, and in most of the cases figured only a single thallus is shown to be present. As von Minden points out, this fungus has affinities with *Entophlyctis*, and in any case it would seem better placed with forms in which the mycelium is reduced to rhizoids (Rhizidiaceae), rather than in the Hyphochytriaceae or Cladochytriaceae where *Catenaria* belongs.

Hitherto the known hosts of *Catenaria* have been mainly nematode worms, though rotifer eggs and the cysts of infusoria also appear to be susceptible, and, as mentioned above, Dangeard obtained thalli in *Nitella*. The form described below occurs in the eggs of the trematode worm *Fasciola hepatica*, the well-known liver-fluke of sheep, and was received from Professor Bayley Butler, of University College, Dublin, in November, 1926. Professor Butler and Mr. Buckley have since published (3) a detailed account of the circumstances which led to its detection, and of the life-history, mode of infection, and development of the fungus in the eggs of the fluke.

The earliest stage observed is shown in Fig. 13 and was exceedingly difficult to make out in the dense protoplasm of the egg. The thallus consists of a main axis, swollen irregularly in places and with a tendency to branch, from which arise a number of very thin-walled rhizoids, given off mainly from the swollen places but also from the unswollen parts. These

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rhizoids. × 225. 5. An egg containing only two connected sporangia. × 225. 6. Various types of short tubes of discharge. × 500. 7. Two sporangia with exceptionally long tubes of discharge, the longest being 67 by 6 μ. × 375. 8. A discharged sporangium with numerous rhizoids. × 430. 9. Dichotomous branching, with sporangial formation in the branches. × 225. 10. A sporangium during discharge, showing zoospores elongated during and after exit and with a long posterior cilium. × 430. 11. Zoospores soon after liberation. × 500. 12. Zoospores coming to rest, showing the two refringent bodies visible at this stage, one apparently joined to the cilium. × 500. 13. Earliest stage of the thallus seen, showing a main recurved axis formed by a rather wide hypha with irregular swellings, and with a single blunt branch at the top. All the other branches are rhizoids with exceedingly thin walls. No septa were visible at this stage. × 225. 14. Later stage. Thallus becoming septate. × 225. 15. A large rhizoid with blunt branches, the ends of which could not be traced. × 500. 16. An egg containing two groups of sporangia, with no fungal elements visible in the intervening space. Isthmuses connecting the sporangia were not visible, but a few rhizoids can be seen. × 225. 17. A similar group to that on the right in the preceding figure. Note the regularly flask-shaped sporangia, with small tubes of discharge. A few rhizoids can be seen (cf. Dangeard's (5), Fig. 12). × 225. 18. An extreme case of multiple sporangial formation, the egg being almost filled with crowded sporangia. × 225. 19. Surface view of egg similar to that in Fig. 18, showing the holes representing the points of emergence of the reduced sporangial beaks. × 225.

rhizoids are either simple or, as in those on the left of the drawing, more or less branched. As in all the material examined, their fine ends could not be made out; as far as they could be traced through the egg contents they were always double contoured, and if they possess terminal branched filaments, as indicated by Dangeard, these must be exceedingly tenuous. The swollen parts figured are doubtless the places which will subsequently develop into sporangia.

At later stages (Fig. 14) the contents of the mycelium become retracted into the swollen parts, which are then cut off by septa and become sporangia. The hyphal segments between the sporangia and also the rhizoids are completely emptied of contents during this process. In many cases a septum can be clearly distinguished cutting off the empty rhizoid from the sporangium, but in others it is equally clear that no septum occurs, as in the large rhizoid arising from an isthmus between two sporangia above the middle of Fig. 1. The rhizoids themselves are without septa (Fig. 15). The sporangia in each thallus often mature at approximately the same time. Thus in Fig. 2 the sporangium with an open tube of discharge had just liberated its zoospores, while the others were on the point of discharging, as could be judged from the condition of the apex of their tubes. The cases figured in Figs. 1 to 5 were selected as showing thalli in which the connexions between the sporangia could be made out clearly. In most cases, however, there are so many sporangia at different depths in the egg that it is not possible to see how they are joined. It was not without considerable search that a few instances were found in which Dangeard's statement that the thallus is dichotomously branched could be confirmed. That this occurs is clearly shown in Figs. 9 and 14, and it may be a more general rule than appears from the drawings, which, as stated, illustrate comparatively simple forms. Very few cases were seen in which the egg contained only one sporangium as in Fig. 4, and not a large number in which the course of a single thallus could be followed throughout the egg. On the other hand, cases like those shown in Figs. 16 to 18 were common. In these an attempt has been made to show only a single optical section; there were other layers of sporangia above and below; and it proved impossible to form any idea of the number of separate thalli concerned in these complex masses, or whether the individual sporangia were united to one another. Dangeard's warning as regards the danger of taking such conditions for species of *Olpidium* or allied forms is fully justified by many of the appearances seen.

The mature sporangium forms a single tube of discharge which may arise from any part of the body, not necessarily, though usually, that nearest the surface of the egg. In a good many cases the tube emerges through the operculum (Fig. 3), which appears to be lifted up by the pressure of the fungus. When the egg wall is pierced, the tube is appreciably narrowed where it passes through the wall, but it swells up just outside and

secretes around itself a thick membrane which becomes thinner towards the apex of the tube. The latter is rounded and before it opens becomes very clear and refringent. Figs. 6 and 7 show some of the types of beak seen, those in Fig. 7 being unusually long, though even longer ones sometimes occur. When sporangia are very numerous the beaks may be small (Fig. 17), and a surface view shows little more than a series of holes in the egg-wall (Fig. 19). The zoospores begin movement within the sporangium, but owing to pressure this is not active until part of the mass has escaped. When only a few spores are left the cilia are sometimes clearly visible, as stated by Dangeard. Those that fail to emerge swim about actively inside the sporangium for a long time, sometimes pausing or creeping around the walls by a distinctly amoeboid movement and then again resuming the actively swimming motion. During exit the spore is elongated and the long posterior cilium can often be seen engaged with the next following spore as if aiding its exit (Fig. 10). In all the cases seen the zoospores emerged one by one and swam away actively as soon as the cilium was free, but Butler and Buckley have seen the first ones escape in a mass and form a ball in front of the opening, held together for a short time by mucus, as described in the earlier accounts. By adding very dilute iodine it is possible to slow down the motion sufficiently to be able to follow it easily. The free swimming movement is smooth, not jerky or zigzag as in so many Chytridiaceae, and lasts for a variable time. Then the spore slows up, gradually becomes more and more rounded, and comes to rest, with the cilium still visible for some time, though shortened. At this period the presence of two refractive bodies in the zoospore is clearly visible (Fig. 12), one being elongated, and, in favourable positions, appearing to be joined to the cilium by a fine line; the other, oval or round and smaller. Germination was not observed, but it and the mode of entry into the egg are fully described in Butler and Buckley's paper.

The dimensions of the fungus are very variable. The sporangia may be as little as 18 μ or even less in diameter, while large ones are up to 90 μ long by 36 μ broad. The tubes of discharge vary from a few μ to at least 80 μ in length by 3 to 6 μ in breadth, and are persistent. The zoospores measure 6·5 to 7·5 μ by 4 to 4·5 μ and are irregularly oval in shape. As in all previous records of *Catenaria*, resting spores were not observed. Sporangia up to 130 by 50 μ and beaks of about 500 μ in length have been observed by Butler and Buckley.

It will be seen that the dimensions of the liver-fluke parasite are in excess of those given by Dangeard and greatly exceed those of Sorokin's fungus. The latter is stated to have zoospores measuring only 1·5 to 2 μ, and Villot states that those of the form parasitic on *Gordius* are 2 μ in diameter. Both these authors describe them as round. On the other hand, Dangeard's species has zoospores up to about 5·5 by 3·4 μ, according to the

figures. In its other dimensions too, Dangeard's fungus is intermediate between those described by Sorokin and in the present note, while that described by Constantineanu is somewhat bigger than Dangeard's. Doubt has already been expressed by Fischer (Rabenhorst's 'Kryptogamenflora', i. 4, p. 144, 1892) whether all the forms on the different substrata mentioned by Sorokin and Dangeard really belong to a single species, while von Minden (loc. cit.) specially refers to the large size of Constantineanu's form. From the observations on the liver-fluke egg parasite, however, it is evident that sporangial dimensions are of no real morphological value in *Catenaria*. The discrepancies in the size and shape of the zoospores are more difficult to reconcile. Villot's observations go to support those of Sorokin, whereas Dangeard's are nearer those here recorded. It is scarcely possible on present knowledge to separate Dangeard's species from that on the liver-fluke, and until the genus is better known it seems advisable to follow his example in disregarding differences in size and to refer this further form to Sorokin's original species. Dangeard included the genus *Catenaria* in the Ancylistaceae, but both Fischer and von Minden place it near *Cladochytrium* in the Cladochytriaceae. Until the resting spores are discovered this seems the best course to adopt.

One other point deserves mention. Amongst the fungi referred to the Ancylistaceae, there are some provided with uniciliate zoospores that have been a cause of difficulty to those who attach importance to ciliation as an indication of natural affinity. *Achlyogeton entophytum* is the chief of these. Sorokin, however, has already mentioned the resemblance between this genus and *Catenaria*: he described as *A.? rostratum* (the only other species known) a form so like the latter genus that Dangeard has queried their identity. If rhizoids have been overlooked in *Achlyogeton* (and the difficulty of seeing them is emphasized above), this genus would be better placed alongside *Catenaria*. Similarly in the genus *Mitochytridium*, where rhizoids have been seen (7), the fact that the whole of the rest of the thallus becomes a single sporangium is, since it sometimes occurs in *Catenaria* (Fig. 4), no reason for separating this fungus from the Cladochytriaceae, where it would seem properly to belong.

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PRESIDENTIAL ADDRESS.

By E. J. Butler, C.I.E., F.R.S.

THE DEVELOPMENT OF ECONOMIC MYCOLOGY IN THE EMPIRE OVERSEAS.

deavouring to sketch the rise and development of economic
ology in the more distant parts of the Empire, the field to
vered is so vast that little more than a bare outline can be
npted. Much that one would have liked to treat in some
l has to be merely mentioned, and many matters of interest
be omitted. I have also had to impose some limitations
y theme or it would become altogether unwieldy, and if I
ttle of the English mycologists, Berkeley, Broome, Cooke,
e, and others who did so much for the early study of the
ous fungi of the Empire, it is not that I am unmindful of
labours. Their work, however, was done in England, and
e endeavoured to confine myself, so far as possible, to the
actually done in the Dominions and Colonies themselves.
om the middle of the last century, there was a rapid growth
preciation of the injury done to living plants by parasitic
. The calamity of the potato blight in the years following
focussed attention on the terrible potentialities for harm
ese organisms, and when from 1869 onwards the coffee
ations of Ceylon, on which the prosperity of that Colony
ly depended, were swept by disease, the lessons of the
o blight epidemic were still fresh in men's minds. Never-
ss, it was more than ten years before the appeals of the
n planters for scientific assistance were met by the decision
nd out a special investigator from home. Harry Marshall
, whose biological training was begun by Huxley at
Kensington and completed under Sachs at Würzburg,
appointed Government Cryptogamist for Ceylon in 1880,
ent out on a temporary mission to study the coffee leaf
e caused by *Hemileia vastatrix*, which by that time had
d a loss of some £15,000,000. He remained only two years,
n that brief period carried out one of the most complete,
was the first, of the detailed studies of a disease of a
cal plantation crop caused by a fungus.
Mr (afterwards Sir) Arthur E. Shipley was sent out
special mission by the Colonial Office to investigate a
t onions in Bermuda, the cause of which in a short time
dated as the fungal parasite, *Peronospora Schleideni*.
years later the Ceylon planters were again faced with a
s disease in one of the crops—cacao—that had replaced

the now abandoned coffee. This time they took matters into their own hands and secured a scientific adviser for themselves. John Bennett Carruthers, the son of William Carruthers, the Keeper of Botany at the Natural History Museum, was appointed Cryptogamist to the Ceylon Planters' Association in 1897 and remained in Ceylon until 1899, a period during which he published a series of reports on cacao canker.

By this time, however, it was abundantly evident that these short tours by specially sent out men did not meet the needs of the rapidly growing agricultural industries of the overseas Empire, and as each Dependency founded a Government agricultural department the appointment of officers for the study of fungous diseases of plants became more and more common.

In this development Australia led the way. In 1890 Daniel McAlpine, who had arrived in Melbourne from Scotland six years previously to take up a lectureship in biology, was appointed Vegetable Pathologist to the Department of Agriculture, Victoria. This and the almost coincident appointment of Cobb to a similar post in New South Wales were, so far as I have been able to ascertain, the first whole-time appointments to permanent posts in applied mycology in the Government service of any Dominion or Colony. McAlpine, who is still living in retirement near Melbourne, was in all respects fitted to be a pioneer in the science of phytopathology in the daughter nations of the Empire. He had studied biology under Huxley and botany under Thiselton-Dyer at the Royal College of Science in London, before becoming a lecturer at the Heriot Watt College, Edinburgh, in 1877. Commencing work at a time when the influence of the great European masters, de Bary, the Tulasnes, Berkeley, and others, who had thrown such a flood of light on the causation of plant diseases through their studies of the parasites, was supreme, he built his foundations on a systematic basis. One of his first preoccupations was to compile a systematic census of Australian fungi, a work of great labour but a necessary preliminary to the identification of the parasites with which he had to deal. The list of Australian fungi that he published in 1895 was in no sense critical and has required much revision, but at least he learnt how little was known in many groups and how the smaller injurious forms had been neglected. Of the rusts, for instance, there are only eighty-one mentioned in the 1895 list, as against one hundred and sixty in the critical monograph that he published eleven years later. As a mycologist he is best known by this and the companion volume on Australian smuts published in 1910, both models of their kind; but in some thirty other papers he has added to the fungous flora of his adopted country in every group.

the more definitely pathological side his three volumes on diseases of citrus fruits (1899), of stone fruits (1902), and potatoes (1911) are well known, and in a series of papers in the *Journal* of the Department of Agriculture, Victoria, and the Bulletins of the same Department, he has largely contributed to our knowledge of the fungous diseases of most of the crops grown in the temperate parts of Australia.

In 1911 he was put on special duty to investigate the nature and control of bitter pit of apples and in five monumental reports, published between 1912 and 1916, he has provided one of the most complete studies of any plant disease in existence. Whether his theories as to the causation of this condition will stand, it is as yet too soon to say, but they mark an ability to apply physiological considerations to the explanation of disease in plants which gives him a definite rank amongst the leading exponents of the modern school of phytopathology.

Nathaniel Cobb arrived in Australia, after studying in the United States, not long after McAlpine and was appointed as Vegetable Pathologist to the Department of Agriculture, New South Wales, at about the same time as the latter. Though in 1893 he published a *Host and Habitat Index of Australian Fungi*, his interests were more in the study of function than of structure and his aims were to discover what the parasites with which he worked did, rather than what they were. In his pamphlet on *Fungus Diseases of the Sugar-cane*, published in the same year, he gave the first description of the bacterial disease still often known as Cobb's disease of sugar-cane, which has since proved such a serious menace to this crop in Australia, Fiji, Porto Rico and elsewhere. He is also responsible for the name "Bitter Pit" applied to the disease that has attracted so much attention in apple-growing countries, and in his "Letters on the Diseases of Plants," published in the *Agricultural Gazette of New South Wales* from 1897 to 1904, a large amount of information on the economic aspect of fungous diseases in Australia is contained. The campaign against rust in wheat, the lines of which were laid down in a series of annual interstate Conferences from 1891 to 1894 and which stands out as one of the most fruitful instances of the co-operation of scientific and practical men on record, had no more energetic supporter than Cobb. During this period, too, he commenced his studies of the nematode worms that attack plants, to which he became more and more attracted as time went on. In 1905 he was appointed Director of the Division of Pathology and Physiology at the Experiment Station of the Hawaiian Sugar Planters' Association and he remained in Honolulu until called to the Bureau of Plant Industry at Washington. In recent years he has abandoned mycology for

helminthology, but he ranks with McAlpine as one of the founders of plant pathology in the overseas parts of the Empire.

The third of the Australian pioneers was Henry Tryon, who, as Assistant Curator in the Queensland Museum, published a *Report on Insect and Fungus Pests* for the Department of Agriculture, Queensland, in 1887. Tryon was appointed Government Entomologist in the Department of Agriculture in 1894 or 1895 but continued to give advice on fungous diseases and was eventually made Entomologist and Vegetable Pathologist, a post which he still holds. He is responsible for some of the earliest records of destructive diseases, such as plum rust, in Australia, but his preoccupation with his entomological duties prevented his giving much time to mycology or plant pathology.

The other States in the Australian Commonwealth were considerably later in developing but have all provided for mycological and plant pathological investigations in one form or another, usually through specially appointed officers in their Departments of Agriculture. The chief contribution to knowledge of the Tasmanian fungi has been made by Leonard Rodway, the Government Botanist, who is one of the leading systematic mycologists of Australasia. Altogether there are now some fifteen members of the various agricultural departments more or less directly concerned with economic mycology in the Commonwealth, and recently a senior mycologist, Dr B. T. Dickson, has been appointed by the Commonwealth Council of Science and Industry to co-ordinate their work.

In 1908 a Government Bureau of Microbiology was established in New South Wales and for some years a considerable amount of work on plant diseases was carried out by members of its staff, especially T. Harvey Johnston and George P. Darnell-Smith. This ceased when the latter became Biologist to the Department of Agriculture.

Outside the Government departments important work on the fungous flora of the continent has been done by Dr J. Burton Cleland of the medical faculty of Adelaide University who, with E. Cheel, the Curator of the National Herbarium at Sydney, has greatly extended knowledge of the higher fungi of Australia, while Miss Ethel McLennan of Melbourne University is doing the same for the Ascomycetes. In plant pathology, too, the Universities are beginning to play an important part, the recent brilliant elucidation of the nature of bunchy top disease of bananas having been greatly aided by the University of Queensland, while Waterhouse and others of Sydney University have investigated cereal diseases. The Colonial Sugar Refining Company have in D. S. North, their privately employed pathologist, one of the leading sugar-cane pathologists of the day.

In the sister Dominion of New Zealand, Thomas W. Kirk, ho was Government Biologist in the Department of Agriculture m 1893 to 1909, published a number of Leaflets and Bulletins aling in popular language with the common diseases of plants the country. At the same time he collected extensively, and ny of the New Zealand fungi determined in England were tained from him. In 1904 A. H. Cockayne became Assistant logist, and to this designation was added that of Vegetable thologist in 1908. Cockayne continued the series of popular icles on fungous diseases in which he had previously collabo- ed with Kirk, when he succeeded the latter as Biologist in 9. It was not, however, until after the war that a mycologist s appointed, Gordon Herriot Cunningham becoming the first vernment Mycologist in New Zealand. In the brief period ch has since elapsed he has devoted himself with extra- inary energy to the study not only of the plant diseases but of the fungous flora of the Dominion. A series of papers on ous fruit diseases published in New Zealand journals was ceeded by a book on the *Fungous Diseases of Fruit Trees*, lished in 1925, which has gained for the author a high repu- on as a pathologist outside his own country. On the syste- ic side he has produced monographs of the rusts, smuts, ularials, and *Cordyceps* of New Zealand and critical re- ons of a large part of the Gasteromycetes of Australasia, des developmental studies of several species of this group. he present time there are two other officers in the biological ch of the New Zealand Department of Agriculture engaged Cunningham in the study of plant diseases and, in addition, rivately endowed Cawthron Institute of Scientific Research as Mycologist, Dr Kathleen Curtis, whose work on potato disease is well known.

have already mentioned that J. B. Carruthers was Crypto- st to the Ceylon Planters' Association from 1897 to 1899. eturned in 1900 as Government Mycologist and Assistant ctor of the Royal Botanic Gardens, Peradeniya, a post h he held until 1905. During this period he continued his on cacao diseases and commenced the study of those of nd rubber. In 1905 he was appointed the first Director of ewly created Department of Agriculture in the Federated y States and after a vain attempt to combine mycology administration obtained the services of a whole time logist in William Gallagher, in 1907. He was thus the f the line of official mycologists in Ceylon and the Fede- Malay States, to whom so much of our knowledge of the es of tea, rubber, cacao, and coconuts is due.

Ceylon, Carruthers was succeeded as Government Myco-

logist by Thomas Petch, who became Botanist and Mycologist to the Department of Agriculture when that Department was founded some years later. For twenty years Petch laboured in the Ceylon Department, studying the fungous flora of the island and the diseases of the two great plantation crops, tea and rubber, which are the main sources of its prosperity. As a result, there is no single part of the British Empire overseas the fungi of which are better known, while in the two volumes on the diseases of *Hevea* rubber and of the tea plant we have the most complete studies of the pathology of single tropical plantation crops in existence. These form only a part of his work. The series of papers on entomogenous fungi that he has contributed to the pages of the *Transactions* of this Society are too well known to need more than mention, and in numerous other contributions he has dealt with critical questions of the morphology and systematic position of a whole series of tropical fungi.

As the work of the Ceylon mycological section expanded, its staff was enlarged. Two assistant mycologists were added to it after the war, besides a group of trained junior assistants recruited locally and in India; while during the same recent period the development of legislative measures for the control of diseases and pests and the general sanitation of crops led to the appointment of Plant Pests Inspectors in the Agricultural Department, working in close collaboration with the mycological and entomological divisions, and including in their ranks several trained and keen mycologists. This latest development of economic mycological work in the Colonies will be referred to more fully later on.

In 1925 Petch retired from Government Service, being succeeded by William Small from Uganda, and shortly after he was appointed Director of the new Tea Research Institute of Ceylon, an institution controlled largely by the planters themselves. Some years previously the rubber industry had provided for its own special needs by the establishment of a rubber research scheme by the Rubber Growers' Association. Under this scheme the Association employs its own mycologist, independently of the staff of the Department of Agriculture.

We have already seen that Carruthers lost no time in developing a mycological branch in the new Agricultural Department of the Federated Malay States. The history of this branch is, however, widely different from that in Ceylon. The extraordinary wave of prosperity that swept over Malaya and the Dutch East Indies as a result of the tremendous growth in the demand for rubber led to a fierce competition between the industry and Government Departments for the services of com-

nt scientific men. Gallagher, after succeeding Carruthers as ctor in 1909, resigned a year later to become technical ser to a large rubber company in Sumatra, and was followed irector by still a third mycologist in Lewton-Brain, whom hall find was one of the early mycologists in the West Indies. h Bancroft was appointed Mycologist in 1910 and resigned 913. E. Bateson was appointed Assistant Mycologist in 1911 resigned in 1914, being subsequently appointed Mycologist Agricultural Adviser to the Chartered Company of British h Borneo. F. T. Brooks acted as Mycologist with A. Sharples ssistant in 1914, and the latter became chief of the branch afterwards. W. N. C. Belgrave entered in 1914 as assistant, had charge of the branch in Sharples's absence on war ice. From 1911 onwards there were three mycological posts he sanctioned cadre of the Department, but they were om all filled at the same time and there were constant ages of personnel. Since the war Sharples has remained in ge, with two assistants.

hile the Government Department was struggling to meet mycological needs of the Malay Peninsula, the rubber in- ry began to appoint mycologists for its own special require- ts. During the war there were six mycologists working in ate employment for bodies like the Rubber Growers' Asso- on, the Malay Peninsula Agricultural Association, and com- es like the Société Financière des Caoutchoucs. As there little co-operation either with the Government Department mongst themselves, there was much overlapping and waste fort; the recent foundation of the Rubber Research Institute lalaya jointly by the Government and the planters them- es, and with its own staff of mycologists adequate for the ls of the industry with James Weir, formerly of the United es Department of Agriculture, at their head, can be directly ed to this cause.

s an inevitable result of the want of continuity in the work lalaya, the development of economic mycology there has markedly different from that in Ceylon. Each officer when inted found himself confronted with one or another of the essive outbreaks of disease that have afflicted the rubber tations. Attention was focussed as a rule on single problems, the bulk of the work done in Malaya has been the mono- hic study of individual diseases and their control. Each of nycologists mentioned has carried out detailed research into cause and control of one or more of the chief diseases of er, and our knowledge of the pathology of the rubber tree ery largely based on their work. None has had time to rtake a general survey of the parasitic and other fungi of

the country and except for a List of the Fungi of the Malay Peninsula compiled in 1921 by T. F. Chipp, Assistant Director of the Singapore Botanic Gardens, and a few other smaller lists, little has as yet been done to gain a knowledge of the fungi of Malaya. Chipp's list, for instance, contains only seven species each of rusts and smuts and nearly two-thirds of the fungi recorded are Basidiomycetes.

One other development in Malaya deserves mention. This is the formation in 1913 of an Inspection Division of the Department of Agriculture charged with the general supervision of the sanitation, mainly from the point of view of insect pests and fungous diseases, of the plantations. F. W. South, then Mycologist in the Imperial Department of Agriculture in the West Indies, was appointed Chief Inspector, with four assistants, a number subsequently increased to keep pace with the growth of the plantation industry. These officers are responsible for the administration of the various plant pest and disease laws as well as the advising on all matters connected with the routine prevention and control of plant diseases. Similar staffs are now found in several parts of the Empire: Jamaica has no less than eighteen Inspectors of Plant Diseases (mainly for the control of Panama disease of bananas), the Gold Coast has about fourteen Inspectors of Plantations for cacao sanitation, Ceylon has three Inspectors for Plant Pests and Diseases, and there are similar officers elsewhere. It is obvious that the presence of a separate staff for such purposes sets free the research officers from much work of a routine nature.

Mycological development in the West Indies as elsewhere was slow and gave few results of permanent value until the appointment of whole time officers in the agricultural service. The Imperial Department of Agriculture for the West Indies was established in 1898 with Mr, afterwards Sir Daniel, Morris at its head. In the following year Albert Howard went out from Cambridge as Lecturer in Agricultural Science in Barbados, and in 1900 he was appointed acting Mycologist and Agricultural Lecturer in the Imperial Department. In his brief period as a mycologist he worked on the diseases of cacao and sugar-cane, his work on the latter crop (in which he was instrumental in establishing the real cause of the terrible rind disease which between 1895 and 1900 threatened the very existence of the sugar plantations) being best known. In 1902 he left to take up the position of Economic Botanist in the Indian Department of Agriculture and was succeeded by Lawrence Lewton-Brain, who also only spent a few years in the West Indies, leaving in 1905 to join Nathaniel Cobb as Assistant Director in Honolulu, whence, as we have already seen, he went to Malaya to succeed

gher in 1910. Lewton-Brain continued Howard's work on -cane and cacao and also studied cotton diseases. He was ded by Frank Arthur Stockdale, who remained for four before becoming Assistant Director of the Department of ce and Agriculture and Government Botanist, British a, in 1909. Like Lewton-Brain, Stockdale after some work e diseases of coconut and cacao soon abandoned mycology a administrative career, becoming Director of Agriculture uritius in 1912 and in Ceylon in 1916. F. W. South fol- Stockdale as Mycologist and Agricultural Lecturer and im remained for four years before proceeding to Malaya in as already mentioned. South was succeeded by William ll, the post of Agricultural Lecturer ceasing to be combined that of Mycologist from 1915. Between 1913 and 1920 ll carried out a number of important researches on the es of limes, cacao, cotton, and coconuts and when ap- ed in the latter year Assistant Director of the Department riculture in Trinidad and Tobago he continued his studies ant diseases both independently and in collaboration with ccessor, S. F. Ashby. In 1923 he published his book on *Diseases of Crop Plants in the Lesser Antilles*, a work which ecured him a position in the front rank of vegetable logists. In 1926 Nowell was appointed Director of the i Research Institute in East Africa, on its revival after a d of stagnation following its capture from the Germans 15.

hile this development in the Imperial Department was on, some of the more important of the West Indian ds appointed mycologists of their own. In 1909 James n Rorer was made Mycologist to the Board of Agriculture, dad. Rorer came from the United States, where he had ed with Erwin F. Smith at a time when the latter was h preoccupied with the bacterial diseases of plants, and soon after his arrival in Trinidad he described two such ses, the so-called "Moko" disease of bananas and the bud f coconuts. The first of these he attributed to *Bacillus Musae*, rganism which Ashby has recently shown to be identical *Bacterium solanacearum*. In regard to coconut bud rot he ccompanied Smith in 1904 on a visit to Cuba to investigate disease and he accepted the conclusion of Smith and Johnston *B. coli* was the cause, a conclusion not substantiated by equent work. Later on he did much work on the green cardine disease of the sugar-cane frog-hopper insect and on ous fruit diseases. His most important discovery, however, that the cause of cacao canker, which Carruthers, Massee, ard, and others had attributed to a *Nectria*, was in reality

a *Phytophthora*, and he made the first detailed study of one of the several major diseases of tropical crops caused by the organism generally known as *P. Faberi*. In 1911 Rorer published a Preliminary List of Trinidad fungi, the only one of the kind contributed by any of the mycologists in the British West Indies, no doubt because few had remained long enough to get to know the fungi of these islands. In 1917 Rorer left the West Indies to take up private employment as mycologist to an association of cacao growers in Ecuador, being succeeded in Trinidad by F. Stell.

In 1906, the Department of Agriculture in Jamaica appointed Sydney Francis Ashby as Fermentation Chemist and Bacteriologist, to work mainly on the microbiology of the rum fermentation industry of the island. Ashby came from Rothamsted with, like Rorer, a special knowledge of bacteriological technique, which he was afterwards to apply to plant pathogens. In 1912 he was appointed Microbiologist, and the study of plant diseases was added to his previous duties. This post he held until he became Mycologist in the Imperial Department in succession to Nowell in 1921. During this period he made important contributions to knowledge of the diseases of bananas, sugar-cane and coconuts and subsequently, on his appointment as Professor of Mycology in the newly established Imperial College of Tropical Agriculture in Trinidad in 1922, he continued the studies of the genus *Phytophthora* which had developed from his investigations on coconut diseases, and joined Nowell in a detailed study of the curious group of yeast-like fungi associated with fruit diseases of cotton and other plants.

In 1921 the Board and Department of Agriculture, Bermuda, employed the well known American pathologist, H. H. Whetzel, to study the plant diseases of Bermuda during a period of vacation from his duties at Cornell University, and he paid a return visit in 1923. Whetzel's recommendations led to the appointment of Lawrence Ogilvie as Plant Pathologist for Bermuda in 1923. In the same year R. A. Altson was appointed Assistant Botanist and Mycologist to the Department of Science and Agriculture, British Guiana. Besides the Professor of Mycology in the Imperial College there are now, therefore, mycologists in Trinidad, Jamaica, British Guiana, and Bermuda.

Following on the mycological appointments in Ceylon and the West Indies, in 1900 the Government of India decided to appoint a Cryptogamic Botanist mainly for the study of the diseases of plants, and the writer was sent out to India early in 1901. Posted at first to the Botanical Survey of India with headquarters in the Royal Botanic Gardens, Calcutta, he was soon after transferred to the newly founded Imperial Depart-

ment of Agriculture and moved to Dehra Dun. When the Agricultural Research Institute at Pusa was built in 1905 he was appointed a member of its staff, and his designation was changed to Imperial Mycologist in the following year. In 1908 William McRae was appointed Second Imperial Mycologist at Pusa, and on his transfer to the newly established post of Government Mycologist, Madras, he was succeeded by F. J. F. Shaw in 1910. In 1919 a third mycologist was added to the mycological section at Pusa, Jehangir Fardunji Dastur becoming Supernumerary Mycologist after nine years as senior assistant in the section. Some six or seven junior assistants recruited locally were also at work during most of this period.

Later on mycologists were added to the Agricultural Departments of some of the other provinces, as well as professors of mycology in some of the Agricultural Colleges, mostly locally recruited men, trained at Pusa. The Indian Cotton Committee have a special mycologist for cotton. The Departments of Agriculture in some of the native States have also appointed similar officers. Leslie Coleman went out from Canada, after a period of study in Washington and Berlin, as Mycologist and Entomologist to the Government of Mysore in 1908, and carried out a series of valuable researches on diseases of areca palms, coffee, and sandal-wood, maintaining his interest in plant pathology after his appointment as Director of Agriculture in Mysore some years later.

Independently of the Government Departments the great tea industry of India founded a scientific department in 1900, and several of the early scientific officers of this department concerned themselves with the diseases of the tea bush. In the *Pests and Blights of the Tea Plant*, George Watt and Harold H. Mann included most of what was known on the subject up to 1903. It was not until 1911, however, that a mycologist was appointed, A. C. Tunstall taking up the post which he still holds. More recently H. Ashplant became Mycologist and Rubber Specialist to the United Planters' Association of Southern India.

India was the first of the tropical Dependencies to take up seriously the study of the parasitic fungi on, and the disease of, the crops grown by the people themselves, as distinct from the plantation crops grown under white supervision mainly for export. In the course of this work the necessity for gaining a knowledge of the fungous flora of the country became apparent and large collections were made by the Pusa staff and have been in great part worked out in collaboration with the Sydows of Berlin. Two earlier Indian mycologists had paved the way for this work. In a remarkable series of papers, published in the

Scientific Memoirs by Medical Officers of the Army of India and elsewhere from 1886 to 1892, A. Barclay contributed not only to a knowledge of the Uredinaceae of India but to the biology of this group as a whole. D. D. Cunningham, also an Indian Medical Officer, published between 1875 and 1897, a small but important series of memoirs on the genus *Choanephora* and on several other interesting fungi that cause plant diseases.

On the pathological side the staff at Pusa and in the Indian Provinces carried out studies on the diseases of cereals, pulses, root crops, oil seeds, drug and spice crops, fibres, palms and sugar-cane. The diseases of tropical fruits and forest trees have not yet been intensively studied either in India or in the other tropical Dependencies, while those of tea, rubber and coffee, the chief Indian plantation crops, have been dealt with mainly by the mycologists in private employ and by those of some of the native States, such as Mysore.

At the present time there are about a dozen mycologists, with a large number of assistants, at work in India, a number all too few for the requirements of the country.

In South Africa the first mycologist appointed was Illtyd Buller Pole Evans who, after a training under Marshall Ward, went as Mycologist to the Transvaal Government in 1905. When the Union was established he became chief of the Division of Plant Pathology and Mycology of the Union Department of Agriculture in 1911, and two years later chief of the Division of Botany and Plant Pathology. The establishment of this fine mycological centre is due to his unceasing pressure between 1907 and 1911 for a properly equipped central mycological laboratory at Pretoria.

In a country which was the birthplace of the father of mycology, Persoon, and where a succession of keen collectors from many nations had worked, it was fitting that Pole Evans should have commenced to study the indigenous fungi of South Africa at an early stage in his career, and should have been mainly instrumental in the formation of what is probably one of the best herbaria of fungi in any of the overseas parts of the Empire. A considerable part of his collections were worked out by the Sydows in Berlin, but the bulk are being dealt with locally. He was fortunate in his assistants and colleagues: Miss Ethel M. Doidge and Paul van der Bijl, who joined his staff shortly before the Union was established, and Miss Averil M. Bottomley, who was appointed in 1913, have all added extensively to the fungi known from South Africa. He himself paid particular attention to the rusts, in addition to a series of investigations on important plant diseases. As his administrative duties increased, Dr Doidge, who became assistant chief of the

division, continued the more purely mycological work. Her monographic studies of the Ascomycetes, especially parasitic and epiphytic forms, are outstanding examples of the monographic treatment of peculiarly difficult groups. On the pathological side Dr Doidge has carried out a series of detailed studies of the bacterial diseases of plants in South Africa, having worked on some fifteen of these, of which four had previously been undescribed. Van der Bijl specialised in the study of the Polyporaceae of the country until he was appointed Mycologist in charge of the Natal Herbarium in 1917, when he took up the sugar-cane diseases that are of such vital importance to Natal, and worked also on various other phytopathological problems. On his transfer as Professor of Mycology in the University of Stellenbosch in 1921 he resumed his earlier interest in the Polyporaceae, of which he has published several lists, while more recently he has taken up the smuts. Two other mycologists have since joined the Division, more especially for the study of the diseases of cotton, tobacco and fruit.

In addition to the central Department, two of the Colonies in the Union have mycological officers. Victor A. Putterill was appointed Government Mycologist, Cape Colony, in 1920, after a period at Pretoria, and his work on fruit diseases is still being pursued in his recent post as chief Government Fruit Inspector at Cape Town. Van der Bijl was succeeded in Natal by H. H. Storey, whose work on the virus diseases of sugar-cane, maize and groundnut will be familiar to many pathologists. Counting the chiefs of the Division at Pretoria there are seven official mycologists in the Union, besides several university and agricultural college professors with mycological interests.

Rhodesia, which though not belonging to the Union may be considered here, has recently appointed a mycologist, J. C. Hopkins, after a considerable amount of collecting and some pathological work had been done by the Government Botanist, F. J. Eyles.

Canada was late in forming a purely mycological staff and has been influenced by developments in the United States in severely directing the work of the Government officers to the practical aspects of plant pathology, a science which in North America has become more and more divorced from mycology proper.

Little mycological work, even on the applied side, had been done when Hans T. Güssow, a native of Breslau who had been an assistant to William Carruthers, consulting botanist to the Royal Agricultural Society of England, was appointed chief of the newly established botanical branch of the Division of Experimental Farms in the Canadian Depar ment of Agriculture

in 1909, with the title of Dominion Botanist. At first he had only a single assistant, and development was slow until the establishment of the field laboratories of plant pathology, of which there are now seven. The first of these was opened at St Catherines, Ontario, in 1912 and others followed in New Brunswick and Prince Edward Island in 1915, in Manitoba and Saskatchewan in 1917, in British Columbia in 1921, and in Nova Scotia and Quebec more recently. That in Manitoba has been absorbed into the newly established Dominion Rust Research Laboratory at Winnipeg, a very fine and up-to-date institution where there is a staff of seven pathologists engaged in the study of cereal diseases under the leadership of D. L. Bailey. At the Central Experimental Farm in Ottawa, the Dominion Botanist has a staff of five pathologists, some of whom are in charge of special branches such as forest pathology, bacterial diseases, and general mycology and plant disease survey.

Besides these officers of the Dominion Department, two of the Provinces, namely British Columbia and Manitoba, have each a Provincial Plant Pathologist, and the Canadian Forest Products Laboratory at Montreal has two pathologists for the study of timber decay. There are altogether more than thirty official plant pathologists employed in the investigation of the cause and control of plant diseases in Canada at the present time.

It is impossible to give an adequate survey in a short space of the field of work covered by such a large number. Güssow himself made valuable contributions to the science of plant pathology in several scientific papers and in the bulletins and annual reports of the Department over a period of years. J. W. Eastham, who was the first scientific assistant on the Dominion Botanist's staff that specialised in plant diseases and who is now Provincial Plant Pathologist in British Columbia, has worked chiefly on fruit diseases, as did Rankin, Hockey, Berkeley and others; Fraser, Bailey and Miss Newton have greatly contributed to knowledge of the biology and control of Canadian cereal diseases; Murphy and Bisby have worked on potato diseases; McCallum on tree diseases.

In the study of the fungous flora of Canada it cannot be said that the official plant pathologists have played a great part. Fraser and Connors have monographed the rusts of the Prairie Provinces and Bisby has published a list of Manitoba fungi in collaboration with Professor Buller.

Even amongst the non-official workers in the Universities and elsewhere systematic mycology has been largely neglected. Faull and Bell have made systematic studies of some forest tree rusts, but the former is better known from his pathological and cytological work and most of the other academic workers have

onfined their mycological interests to the fungi as producers of disease. Probably the largest contribution to knowledge of he Canadian fungi has been made by an amateur, Dr John Dearness, whose collections and descriptions are well known to ll students of North American fungi.

As a contributor to knowledge of the biology of the fungi in ts widest and most varied aspects Arthur H. R. Buller, Professor of Botany in the University of Manitoba, stands alone. Going out from the University of Birmingham in 1904, he published the first volume of his *Researches on Fungi* in 1909 and n the subsequent volumes of this work and in many single papers he has not ceased to elucidate the inner secrets of the life of these plants. Both in time and in the importance of his contribution, his rank amongst the pioneers of mycological development in the British Empire overseas is secure.

In British tropical Africa the first official mycologist was Charles Ogilvie Farquharson who, after training at Aberdeen and work in the Jodrell Laboratory, Kew, went out as Mycologist in the Agricultural Department of Southern Nigeria in 1911. His tragic death by drowning at sea, when homeward bound during the last few weeks of the war, cut short a career of unusual promise. Walter John Dowson took up the position of Mycologist in the Department of Agriculture, British East Africa (now Kenya) in April, 1913. A month earlier William Small started work in the neighbouring colony of Uganda as Government Botanist and at once concentrated his attention on the diseases of plants to which he continued to give most of his time until he became officially the Mycologist to the Department of Agriculture in 1920. In the Gold Coast, Robert Hugh Bunting was appointed Government Mycologist in 1915, in addition to being Assistant Director of the Department of Agriculture. These were the four pioneers of the development of economic mycology in tropical Africa. Further appointments were made in rapid succession after the war, until at present there are two mycologists in the Gold Coast, two in Nigeria, and one each in Kenya, Uganda, Tanganyika, and Sierra Leone. Zanzibar also employed one for special work on clove diseases during a period of six or seven years.

The work in each of these Colonies has been naturally determined mainly by the nature of the local agriculture. In Kenya, Dowson and his successor, McDonald, have done much in the study of the diseases of the coffee bush; in the Gold Coast, Bunting and Dade have concentrated chiefly on cacao diseases; and in Uganda, with its more diverse agricultural interests, Small worked on diseases of coffee, cereals, and various native crops. In all parts of British tropical Africa plant diseases are

being intensively studied at present, but it will be many years before they are adequately known and the full extent of their depredations realised. The first general handbook of plant diseases in any part of this vast area that has appeared is Bunting and Dade's *Gold Coast Plant Diseases* published in 1924. Apart from the parasites, relatively little attention has been given to the fungous flora, though Farquharson and MacFie in Nigeria, Bunting in the Gold Coast, Dummer, Maitland, and Small in Uganda, and Maitland and Dowson in Kenya have made considerable collections, some of which have been worked out at Kew. There is no doubt that our relative ignorance of the fungi of tropical Africa is a serious handicap to economic mycological work in that part of the Empire.

Though no longer a part of the Empire, Egypt deserves mention here, if only on account of the pioneering work on cotton diseases carried out by W. Lawrence Balls between 1904 and 1913, when he was botanist to the Khedivial Agricultural Society and the Department of Agriculture. A whole time mycologist, however, was not appointed until after the war, when H. R. Briton-Jones was given the post.

A combined post of Botanist and Mycologist was created in the Mauritius Department of Agriculture in 1922 when E. F. S. Shepherd from Macdonald College, Quebec, was appointed to it. Even in the far Pacific, a Government Mycologist, J. G. C. Campbell, is at work in the Fiji Islands.

In the Dominions, Colonies and Protectorates there are now about a hundred mycologists and plant pathologists in Government Service, counting only those whose duties include some form of research or experimental work. As I have shown, this development has come about almost entirely since the beginning of the present century. It has resulted from the tremendous development that has taken place during the same time in the demand for the agricultural produce of these countries. All these staffs are engaged in the study or control of plant diseases. The value of the crops whose health they have to safeguard is enormous. The agricultural exports of the Colonies alone, excluding the Dominions and India, were worth £171,000,000 in 1926. Rubber was exported, chiefly from Malaya and Ceylon, to a value of not far short of £100,000,000. In tea, rubber and coconut produce Ceylon exported over £33,000,000 worth in 1925. In the same year India exported over £20,000,000 worth of tea, about £65,000,000 worth of raw and manufactured jute and over £60,000,000 worth of raw cotton, all grown in the country. Of crops mainly used in the country there are

30,000,000 acres under rice in India, and 30,000,000 (yielding 60,000,000 tons) under wheat. The Gold Coast supplies more than half the world's requirements of cocoa, exporting £8,000,000 to £10,000,000 worth annually.

Figures like these mean little, however, unless it is remembered that the agricultural industry is almost the only one in many of the Colonies. Their whole prosperity is bound up with it and they are threatened with ruin when an epidemic disease breaks out in one of their main crops. The disasters that overtook Ceylon when its coffee industry was destroyed by leaf disease, and the West Indies when rind disease of sugar-cane swept the old varieties of cane out of existence, may be repeated at any time and indeed have been repeated on a smaller scale quite recently, when withertip of limes destroyed 90 per cent. of the crop on some estates in Dominica in a year or two after its first appearance and forced the island to appeal to the home Government for relief. That such outbreaks are not more frequent can be attributed in great measure, I think, to the work of the economic mycologists. No one can doubt, who knows the facts, that the rubber industry of Malaya has been saved not once but several times by the control measures devised by the local mycologists against such diseases as pink disease and mouldy rot, and if the present uneasiness about the spread of the rubber *Oidium* becomes justified, as it well may, a greater effort than any in the past will be required to protect this enormous industry.

It is difficult for people in this country to realise why disease becomes so much more dangerous a factor in the agriculture of these newly developing countries than it is at home. The first great difference between the conditions in the two cases is the tendency to plant large areas continuously or almost so under the same crop. This is true not only in permanent plantation crops proper, like tea and rubber, where one may pass for miles on end through the crop, broken only occasionally by a belt of jungle, but it is even more true for the cereal areas of Canada and Australia. Parasites find optimum conditions for their depredations under such circumstances. A spore liberated from an infected plant stands a good chance of landing on a susceptible host, and every individual plant undergoes such an intensive bombardment that the weak spots in its defences are likely to be attained. Then, a low cost of production is essential if the produce has to be sent long distances to its market, and it is not possible to pay the same attention to the general sanitation of the plants as is given under more intensive agricultural conditions. And, finally, the crops are often relatively new introductions and there has been as yet no time for the

action of the process of selection of disease-resistant varieties that has consciously or unconsciously been going on in the older countries for generations. The economic mycologist overseas is thus continually liable to be faced with outbreaks of disease of a much more severe type than are usually encountered at home, and that can only be paralleled by attacks of newly introduced diseases, such as American gooseberry mildew or wart disease of potatoes, in Britain. He is fortunate, indeed, when he has to deal with a known parasite with the means of control already worked out.

On the human side, too, the work is of not less absorbing interest. One must have experienced it, to know what it means to find a native village that has specialised in the growth of some particular crop or variety faced with the total loss of that crop by an unknown disease. When large districts are similarly affected the matter soon comes to light, but small areas may suffer in silence. When one can help these people one is fortunate, but failure owing to the obscurity of the disease or want of time for working it out is still too frequent. An equally painful impression is left when the white planter who has, perhaps, put all he possesses into a tea or coffee plantation in one of the colonies finds fifty or seventy-five per cent. of his bushes suffering from a disease on which not enough work has yet been done to allow of adequate control measures being recommended. Such cases make the observer feel that no increase in the numbers of men engaged in the struggle against plant disease in these mainly agricultural countries can be too rapid; that a constant supply of new men trained in their study is wanted; and that there is no field of mycology which better repays cultivation than that worked by the mycological and plant pathological departments in the overseas parts of the British Empire.

STRAINS AND TAXONOMY OF PHYTOPHTHORA PALMIVORA BUTLER (P. FABERI MAUBL.).

By S. F. Ashby, Mycologist, Imperial Bureau of Mycology.

(With nine Text-figures.)

OBSERVATIONS by economic mycologists during the last ten years especially, have shown that the omnivorous tropical species under consideration exists as a number of morphologically distinguishable and in some instances biologically distinguishable strains on a wide range of cultivated plants. It has been recognised as causing a fruit rot and patch canker of cacao (1) in

The Delimitation of Species of Fungi on Physiological Grounds

E. J. BUTLER

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THE DELIMITATION OF SPECIES OF FUNGI ON PHYSIOLOGICAL GROUNDS[1]

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Mycologists who are accustomed to the study of those fungi that are obligate parasites — the rusts, powdery mildews, many of the downy mildews, and so on — know how great is the temptation and how frequently it is yielded to, to distinguish species by their ability to attack particular host plants and their inability to grow on others, rather than by any morphological character.

Those too who have worked extensively with cultures of fungi on artificial media, who have studied groups in which cultural characters have come to be given importance in classification, know equally how common it is to found species on some particular growth character or even on biological peculiarities, such as capacity for fermenting certain sugars, discoloring certain media, and the like.

Examples of these tendencies are easily given and could be multiplied. The study of the rust fungi has developed almost from the beginning with the tendency, strongly marked, to distinguish species according to their hosts. When a rust is found on a particular host, search is made amongst the species of the genus already recorded on the same host, or perhaps on a few allied species, and if none is found to agree, it is frequently regarded there and then as a new species. Indeed Fischer in "Die Uredineen der Schweiz" regards all rusts as distinct if they differ in the genus of plants to which they are restricted. In many cases in which a species was originally described as occurring on a number of hosts, there has been a disposition to separate it into several species each confined to a particular host genus (for example, *Puccinia rubigo-vera*, *P. lolii*, *Uromyces trifolii*). A further step has been attempted: when a species has a number of strains confined to different varieties of the same host plant, for example, *Puccinia graminis* on wheat, suggestions have been made to split it into several species, some of which would have somewhat distinct morphological characters.

In the smuts the distinction by hosts is almost complete. In spite of the fact that one was aware in a general way that *Ustilago tritici* and *U. nuda* were morphologically similar, it comes as a surprise when one finds the loose smuts of wheat and barley united into a single species *U. tritici*, as Cunningham ('24) has done in his recent monographic treatment of the smuts of New Zealand.

The Erysiphaceae as dealt with in Salmon's monograph ('00) form a good expression of the opposite tendency to that which has been shown on the whole

[1] Presented before the International Congress of Plant Sciences, Section of Mycology, Ithaca, New York, Aug. 17, 1926.

in the rusts and smuts. The 160 species that are recorded in Saccardo's "Sylloge" as having been named up to that date were reduced to 49 and 11 varieties, in spite of the fact, established largely by Salmon himself, but also by Neger and Marchal, that many of these species contain specialized forms restricted to one or a few hosts. Though Blumer ('21–'25) has shown that certain of these forms can be distinguished by morphological characters, the size of the conidia differing on different hosts, he has been cautious in giving specific rank to any but rather extreme cases. Jørstad ('25) in his recent monograph of Norwegian Erysiphaceae follows Salmon, except that certain of the latter's varieties are considered to be distinct species. A study of the synonymy, and of the range of the characters on the different hosts and their gradation into one another, as set out in Salmon's monograph, shows clearly the disadvantage that would result, in this group at least, from distinguishing the species according to the hosts on which they occur.

Coming to the downy mildews, the genus *Peronospora*, as represented in Saccardo up to 1910, contained 113 species, a good many of which were probably synonyms. Gäumann's monograph ('23) lists 267, of which 146 have Gäumann's name attached. The single species formerly recognized as a crucifer parasite, *P. parasitica* (Pers.) Fr. is divided into 54, of which only five were recognized by any earlier writer as having specific rank. These are not distinguished by oospore characters, nor can they be recognized by those of the conidiophore, since of the 15 types of the latter distinguished by the author, all intermediate stages can be traced. A biometrical study of the conidial dimensions shows that, while each of Gäumann's species differs to some extent from the others, the peak of the curves for length and breadth gives only a fraction of a micron difference in many cases, while the quotient of length by breadth is identical or very close also in many cases, giving, for instance 1·16 in five, 1·15 in four, 1·14 in four, and so on. Since the age of the conidia affects this figure, as well as the other data, it is evident that little can be gained by attempting to separate the species on this basis. From these morphological characters alone, Gäumann ('16–'18) considers that one can do no more than place a given example of the genus in the "collective species" to which it belongs, in this case *P. parasitica* of the older authors, further separation into the individual species requiring, as the only constant and indisputable means of distinction, a determination of the physiological selection of host plants. It is only on this last basis that his 54 species can be identified, for though in the diagnosis of each the morphological characters are also given, it is evident that without knowledge of the host, identification would be impossible in a large proportion of them. How widely this conception departs from that in Salmon's Erysiphaceae is at once evident. If generally accepted, it would be difficult to deny specific rank to the numerous races of the fungus still usually known as *Puccinia graminis*. That it is not likely to be generally accepted, even in his own country, is evident from Fischer's ('17) correlation of Gäumann's forms with Johannsen's pure lines of beans, and his relegation of them to the group of "kleine Arten" or "formae speciales."

If we turn to the fungi that have been critically studied in artificial cultures, whether facultative parasites or merely saprophytes, we find a similar diversity

of outlook. Westling's monograph ('11) of the green species of *Penicillium* is stated by the author to be a critical study on a morphological basis. Thom also has maintained, on the whole, a morphological criterion for his species. Both attach a certain weight to physiological characters and both insist that the morphological characters should be studied on uniform media. But, as Westling says, from this standpoint to one in which physiological data are given a dominant position in the diagnosis of species is all too great a step. Biourge ('23), dividing the genus on growth characters, has multiplied species in a manner that recalls Gäumann. He states that whereas Saccardo in 1900 had listed only 42 species, with 2 *Citromyces* (now included in *Penicillium*), he himself found over 290 names in existence in literature. In his monograph he accepts 128 species and admits that he has not been able to recognize in his cultures many of the species described by others. Even in the division of his sections he makes use of cultural characters, such as zonation in Petri dish cultures, and, though his species have diagnoses including morphological characters, it is probable that it would be impossible to determine many of them by these alone, without the extensive series of cultural tests that are appended in each case. Like his predecessor Dierckx, cultural characters are given greatest weight, and of the latter's handling of the genus Lindau ('07–'10) remarks: "This does not make for clearness, but only increases the confusion."

An extreme case of the use of biological characters in classification is presented by the genus *Monilia* as recognized by medical workers. Castellani's ('25) method of distinguishing species of these fungi is based primarily on their fermentation of a series of sugars, since, as he puts it, morphological grounds do not suffice for a classification. In the genus *Beauveria*, including the fungi parasitic on insects formerly known as *Sporotrichum globuliferum*, *Botrytis bassiana*, etc., attempts have been made to separate species by the color they impart to various culture media. As might be anticipated, recent studies by Petch ('26) have shown that this character is inconstant and cannot be relied on. Precisely the same thing is happening in regard to the color and other cultural characters used in the diagnosis of the ringworm fungi, as a result of studies of large numbers of isolations.

Fungi that belong to what are sometimes known as the hemi-saprophytes, that is, species mainly parasitic but capable of a saprophytic life and, therefore, culturable artificially, present several examples of the difficulties of the systematist. The section *elegans* of the genus *Fusarium* contains a number of vascular parasites of all kinds of herbaceous and shrubby plants. In Erwin F. Smith's ('99) classical study of the forms causing wilt of cotton, watermelon, and cowpea, those on the two latter hosts were regarded as varieties of that on the first, on the ground apparently that each was restricted to particular hosts, since it is definitely stated that all are morphologically identical. Other varieties were subsequently added, for example, *F. vasinfectum* var. *pisi*, the cause of the "St. Johanniskrankheit" of the pea in Europe. Later on all these have been given specific rank. If now one examines the morphological characters of many of these *elegans* forms, as shown in Wollenweber's "Fusaria autographice delineata," one

is forced to admit that Smith's earlier view was justified, since the variations shown in the five plates of *F. orthoceras* are greater than the differences between, say, *vasinfectum*, *tracheiphilum*, *niveum*, *oxysporum*, *hyperoxysporum*, and *cubense*. In regard to the last of these, the cause of the Panama disease of bananas, both Brandes and Wollenweber state that it is scarcely different from *F. vasinfectum*. I have had three of these vascular parasites under study in India, the causes of wilt diseases of pigeon pea, cotton, and sesamum. I described ('10) the former as a new species, *F. udum*, mainly on cultural characters. Subsequently I considered the cotton wilt producer of India as distinct both from *F. udum* and from the cause of cotton wilt in America, *F. vasinfectum*. Recent comparative study of the sesamum fungus and of *F. cubense* has failed to show any morphological differences, and the cultural differences that were earlier noted in the case of the other two, both between themselves and from *F. vasinfectum*, have broken down. Even the biological character of host specialization appears to be less fixed than had been believed. Small ('22) found in Uganda a fungus morphologically and physiologically identical with *F. udum* but capable of attacking many other plants besides pigeon pea. Similarly *F. cubense*, as it occurs in the Caribbean, cannot infect *Musa cavendishii*, but a morphologically identical fungus causes a wilt of this species of banana, indistinguishable from Panama disease, in the Canaries. And in Egypt Fahmy (according to letters received from him) has under study three strains of cotton wilt-producing fungi of the *F. vasinfectum* type, one from America which mostly attacks American varieties; one from India restricted to Indian cottons; and one from Egypt chiefly on the Egyptian. Morphological differences between the three strains are very slight, but there are cultural differences. If specilization of parasitism is the guide, there would seem to be as good reason for giving specific rank to the Indian strain, which refuses to attack Egyptian cottons, as to most other species in the *elegans* section, but what is to be done with the Egyptian strain which sometimes attacks the Indian varieties?

In all these cases the problem resolves itself into one of the relative weight to be attached to a certain group of physiological characters in classification. The same general set of physiological characters is responsible for the ability or inability of an obligate parasite to grow on a particular host plant as for a saprophyte to show certain characters in artificial culture. It is largely a question of being able to use particular nutrient materials or of being able to alter substances by enzyme action so that they may be assimilated or at least neutralized. It appears to be extremely doubtful if any more weight should be attached to host specialization in the parasites than to cultural characters in the saprophytes, and if we refuse to except the one in classification it is difficult to justify the use of the other.

There are, indeed, quite evident signs of a revolt against the subdivision of species on biological characters. Thus Cunningham ('23) wishes not only to unite *Ustilago tritici* and *U. nuda*, as mentioned above, but also to restore the old species *Puccinia elymi* to include *P. triticina* with several other rusts now regarded as distinct, and Mains and Jackson ('26) have recently indicated their

sympathy with such a view, while doubting the expediency of altering what has become established usage. Hansford ('25) has argued that the present classification of species in the *elegans* section of *Fusarium* is useless and that it is preferable to regard all those, at least, that he has studied (including quite a large number ordinarily recognised as distinct) as strains of a single species. Small, in a recent study of strains of the genus *Colletotrichum* in Uganda ('26), considers that nearly all those he has encountered on various hosts are but forms of a single morphological species.

We can recognise three distinct tendencies in the classification of fungi of the types under consideration. The older, purely morphological one, as defined by Marshall Ward ('05) who considered that "no species can be accepted as valid until it is capable of definition in morphological terms," a view which is being reverted to by some at the present time; the almost purely biological, as instanced by Gäumann's treatment of *Peronospora*; and that in which both morphology and physiology are taken into account. Is there any direction in which guidance can be sought as to which of these tendencies may best be followed? I think that such guidance can be found in certain evolutionary considerations.

In his presidential address to the Zoology Section of the British Association last year, Regan defined a species as a community or a number of related communities, whose distinctive morphological characters are, in the opinion of a competent systematist, sufficiently definite to entitle it or them to specific rank. No rule is required for the guidance of the systematist in this; convenience may often be the best criterion. He illustrates with a number of striking cases, the view that throughout the evolution of the fishes, changes of structure have been intimately related to, and may even be said to have been determined by, changes of habits. The importance still given to adaptive factors in evolution by competent biologists, as illustrated in this case and also in H. F. Osborn's opening address to the same section in Oxford, ten or twelve days ago, may make us more ready to admit that the mere colonizing of a new host plant by a rust or mildew may lead not only to special biological characters but also to some morphological changes, which may become in time, no matter by what agency, permanent. How else can we explain the slight morphological differences so often noticed in biological races?[2] It is obvious that a stage may arise when it becomes a question whether such adaptive modifications are of sufficient magnitude to justify specific rank. It is here that the "competent systematist" appealed to by Regan has his field. It is assumed that he has a knowledge of the allied species and perhaps an inkling of the form from which they have been derived. Without this, his opinion is valueless. With it, he may say, for instance, that all the races of black rust are sufficiently like each other, and sufficiently different from other rusts,

[2] The absence of a previous host plant, as must often occur when a plurivorous species spreads to a new area, might have a similar influence. Fischer ('13) found a form of *Uromyces caryophyllinus* that in the Wallis (Switzerland) region infected *Saponaria ocymoides* and *Tunica prolifera*, whereas when collected in Baden, where *S. ocymoides* is absent, the spores had lost the power to infect this plant, though *Tunica* was readily attacked. It is, no doubt, in some such way that Fahmy's strains of *Fusarium vasinfectum* originated.

whether those that occur on the same host plants or elsewhere, to be kept within the species *Puccinia graminis*. He may consider that all the downy mildews on the Cruciferae are sufficiently alike to be included in *Peronospora parasitica* or a few (a very few, I think) can be readily recognised and may be regarded as different species. If there were no large range of intermediate forms, as may come about by the suppression of some, it might often be preferable to recognise several species in an allied group. Regan points out that if there were only Englishmen and Hottentots we would probably speak of two species of man. From the evolutionary standpoint it is often a matter of convenience where we draw the specific line, and convenience demands generally a morphological criterion, since it is obviously not convenient to the taxonomist to have to carry out an extensive series of experiments before the proper pigeon hole into which to put an organism can be found. The cerealist or phytopathologist who wishes to know exactly what strain of rust he handles, or the physician in dealing with the ringworm fungi may require to do this, and it may be left to them, as it is left to the agronomist or horticulturist to know the cultivated varieties of the plants with which they deal.

Furthermore, and this applies almost as much to the biological races as to the saprophytes with cultural strains, adaptive characters are liable to fluctuate rather freely, perhaps as a result of changes in the environment. Hosts that are resistant under one set of conditions may become susceptible under another. Even a variation of temperature may sometimes break down resistance to infection, as shown at Madison and elsewhere, and it must not be forgotten that for a parasite the host plant constitutes a large part of its environment. The forms of *Fusarium vasinfectum* in America, India, and Egypt and of *F. cubense* in the West Indies and Canaries might not maintain their host specialization under modified conditions. I do not understand how they could have come into being without plasticity of some sort. Saltants are now known to differ at times from the parent strain in virulence, and there are various way in which a fungus might change its host range. Hence it is difficult to admit that biological specialization alone is a sufficient basis for distinguishing species unless one is prepared to face a large number of cases in which this criterion will not serve.

Still less in the case of moulds like *Penicillium* can cultural characters be given specific weight, though they may bring support to morphological differences. Color and type of growth (aerial mycelium, formation of coremia, pionnotes, etc.) are two characters much used in the cultural identification of species, but they are unstable characters, like most of the others of this category. It is possible to "stretch" the cultural characters of a group of strains of a single organism, such as *Fusarium blackmani*, by controlled modifications of the medium on which they are grown, as has been shown by Brown and Horne ('25–'26). The amount of aerial mycelium, rate of growth, zonation, color, amount of sporulation, septation, and even to some extent size and shape of the spore, can be made such that, if constant, they would denote distinct species; yet all can be derived from a monospore or single hyphal tip culture, intermediate forms exist, and each can be made to resemble the other. F. L. Stevens

('22) dealing with a similar case in the genus *Helminthosporium* points out that it would be possible by biometrical, biological, and cultural criteria to draw up distinctive diagnoses for a number of the races he studied, but he prefers to regard at least the great majority as falling within the Linnean species, *H. sativum*. In some cases it would appear that morphologically good species, readily recognisable as such in nature, can be "stretched" in culture so as to overlap one another. The writer believes that this has occurred in Leonian's ('25) recent cultural study of most of the described species of *Phytophthora*, where some 24 species and strains were united into *P. omnivora*. Cultural study here has led to the view that the size and shape of the fruiting bodies are minor characters, but some, at least, of these fungi can readily be identified as they occur in nature and, indeed, in culture, and if this is so it is a strong argument for keeping them distinct.

Considering these and similar cases in the light of convenience, it appears that it may sometimes be preferable to unite and sometimes to divide. It may be convenient for practical reasons to keep *Ustilago tritici* and *U. nuda* distinct. *Colletotrichum limetticolum* is probably, as Burger ('21) holds, merely a strain of the ubiquitous *C. gloeosporioides*, but it causes the destructive withertip disease of limes, whereas the common forms of *C. gloeosporioides* on *Citrus* are, at most, weakly parasitic, and so it may be kept apart. The disadvantages of multiplying species are so many, however, that we would probably be well advised to regard such cases as entirely exceptional.

If we admit that there is no such thing in nature as a pure, invariable, homogeneous species, that habit can modify the facies of a species so long as it endures, that morphological characters are less readily modified by the environment than physiological, and that convenience is often the best guide where to draw the line between different species, we may get some degree of guidance as to which of the tendencies defined above may best be followed. We will be led, I think, on the whole to prefer the morphological basis of classification and only in special cases to stress biological differences.

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INTERNATIONAL PLANT DISEASE LEGISLATION AS IT AFFECTS THE BRITISH EMPIRE[1]

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I propose to give a very brief survey of conditions in regard to plant import regulations as they affect especially the British Empire and to discuss a few of the general principles on which such regulations are based. Conditions in England are widely different from those in many other parts of the Empire and the difficulties and responsibilities of official plant pathologists in England are much less than in the British Dominions and Colonies. The agricultural industry of England is a relatively unimportant one, compared with the other great national industries, and it is also a very varied one, not dependent on the growing of a few crops on a large scale, as is the case in some of the exporting Dominions and Colonies. There is nothing in England comparable to the rubber-growing industry of Malaya, the tea, jute, and cotton industries in India, or the cocoa industry of the Gold Coast, industries on which a great part of the national prosperity of these countries depends. The Government plant pathologist in such countries has to watch very carefully for possible dangers arising from the introduction of foreign diseases, and may have to take measures of considerable stringency in order to safeguard a very valuable industry and one which often it would be hard, or perhaps impossible, to replace. As it happens these measures do not ordinarily affect England, since most of the crops concerned are grown in the tropics and are not liable to serious danger in regard to importations from temperate zones. But, so long as these considerations have weight—and that is likely to be for a very long time—it is useless to hope to get the British Empire mycologists as a body to endorse any proposal for the abandonment of their powers to impose severe restrictions on the free circulation of living plants. The persons responsible for the safety of the Eastern rubber industry cannot agree to any proposal which would force them to admit living rubber plants from South America and the Caribbean, plants which might bring in the Hevea leaf disease that has prevented the establishment of a plantation rubber industry in those regions. Not only would the mycologists oppose any such suggestion, but the whole rubber industry would be up in arms at once and could prevent the Governments concerned from agreeing. I mention such a case merely to show that no general proposals, on the lines of those of the Rome Convention of 1914, that might make it obligatory to admit living plants even though these be certified free from disease, have any chance of being accepted in the Empire as a whole. Certain total prohibitions will remain in force.

[1] Presented before the International Congress of Plant Sciences, Section of Pathology, Ithaca, New York, Aug. 20, 1926.

Apart from these cases in the tropical Dependencies, which are not as a rule serious impediments to trade and only affect England slightly, there are numerous quarantines and restrictions on plant imports in the temperate Dominions, States, and Colonies. They are based on certain biological facts and principles regarding which there is room for much more information than we possess at present. Both in the British Empire and in the United States the necessity of distinguishing between two types of dissemination, at least as regards fungi and bacteria, is being more and more recognized. There is the problem of dissemination of a parasite within a continuous land area, whether the area is divided up by political boundaries or not, and there is that of discontinuous dissemination, mostly between areas separated from one another by the ocean.

In regard to the former of these, most pests of long standing have already reached all parts of the continuous land area in which they can maintain a footing, and restrictive measures are generally confined to newly-introduced pests which have come from other parts of the world. We do that in England. We do not impose restrictions as a rule in the case of diseases of long standing, since these have reached all parts of the country, but diseases like wart disease of potatoes are subject to quite different treatment. So in the United States and Canada it is chiefly the new diseases that are responsible for internal restrictive legislative measures.

The prevention of the introduction of exotic pests from a distance, mainly from overseas, is a very different matter. Here much exact knowledge is required and is not always readily available. With fungous diseases one has to have clearly in mind the crop which one wishes to protect and then to examine what pests it suffers from in other parts of the world that might be destructive if introduced. Unfortunately one has often to go further and consider what parasites of other plants, be they related to the crop one desires to protect or not, might prove dangerous if introduced. Then one has to consider the powers as a traveller of these parasites. Here we have a point which has been very critically examined of recent years. In 1914–15 I examined the question[2] in the light of the known facts of the spread of a large number of plant diseases whose history could be traced, and came to the conclusion that the powers of long distance dissemination of the parasites had been frequently exaggerated. In most cases spread along trade routes could be clearly traced and spread by natural means without human agency could not. A few years ago Orton and Beattie[3] of the Federal Horticultural Board in the United States stated that no case of long-distance transfer of plant disease by wind had been demonstrated, and that carriage of disease in ballast, except where soil was concerned, could not be definitely traced. In most of the overseas parts of the Empire it is this long distance or discontinuous dissemination that is important. The most frequent agent in such dis-

[2] Butler, E. J. The dissemination of parasitic fungi and international legislation. Mem. Dept. Agric. India. Bot. Ser. 9(1): 1917.

[3] Orton, W. A., and Beattie, R. K. The biological basis of foreign plant quarantines. Phytopathology 13: 295–306. 1923.

semination is man, and the outstanding medium of transfer is living plant material.

That is why the countries that import living plants watch their ports and intercept diseased plants or prevent their arrival.

In examining the steps taken by importing countries we might enquire; (1) are they based on sound biological data; (2) do they secure the objects aimed at; (3) do they go beyond what is necessary or expedient in securing these objects?

In regard to the first of these questions I do not propose to delay. I regard it as fully proven and abundantly illustrated by terrible examples that the main danger from exotic plant parasites lies in the readiness with which they can be introduced on living plant material, while excepting for this method of introduction there is little risk to be feared from them.

With regard to the second point, the adequacy of the measures taken to keep out exotic parasites, this of course varies from country to country as the measures taken, themselves, do. Wart disease of potatoes has been successfully kept out of many parts of the world and it is interesting to note that the first case of this disease in South Africa was brought in before the introduction of legislative measures to exclude it, though not discovered for a number of years. I believe that every part of the British Empire is satisfied that it has, in fact, kept out certain dangerous diseases by taking the necessary precautions, diseases which in the absence of any precautions would have come in.[4]

The answer to the third question, which chiefly affects plant-exporting countries like England, namely, whether the measures taken go beyond what is necessary or expedient to secure the objects aimed at, is not so easy. One finds in some countries a long list of plant diseases to be excluded, in some a shorter list of specified diseases, but, in addition, a regulation prohibiting in general terms the introduction of any diseased plant or of certain categories of plants, for example, nursery stock. It is probable that no plant pathologist would care to certify any consignment of plants as being free from all diseases, whatsoever. If, therefore, such a measure as that in Section II of the Canadian Act, which reads "No pest or disease or plant infested with any pest or disease shall be admitted into Canada," is intended to be taken literally, it would mean practical prohibition. But if it is intended merely to give power to reject any obviously diseased consignment, even when the disease present is one not specially listed, then no objection can be taken to it. Such a general clause might then even help in keeping the specially listed diseases down to a limited number, and actually assist the careful exporter in gaining admission for his consignments.

In countries which list a large number of specified diseases one finds included in this number, sometimes, diseases that are already known to occur in the importing country. I think great care is required in handling the regulations restricting the entry of these. It is, I think, quite right that consignments with a high percentage of disease should be refused, even if the disease be one already

[4] We need only to compare the spread of potato blight with that of wart disease within the British Empire to be convinced of this.

present in the country, but this ought not to apply to consignments containing a few odd cases. To refuse 100,000 rose grafts because 20 or 30 have crown gall—when crown gall is already widely present throughout the country—is indefensible. In the report of a committee of the American Society of Horticultural Science on crown gall inspection, published a few years ago,[5] it is stated that the Committee regards as unjustifiable the rejection of an entire shipment on account of a slight incidence of this disease. What I would like to see in similar cases is the exclusion of crown gall altogether from the listed diseases, and the application of some general section which would allow the inspector to reject an obviously badly galled consignment.

In general, one would like to see the number of kinds of plants prohibited on account of specified diseases kept down to a few really important or dangerous things. I admit that it may be hard to know where to draw the line in continental areas like Australia and Canada. Still Canada has only listed specifically for fungous and bacterial diseases nine kinds of plants, namely potatoes, five-leaved pines, chestnuts, *Ribes*, *Rhamnus*, Barberry, three kinds of coniferous trees liable to attack by *Phomopsis pseudotsugae*, peaches from certain parts of the United States, and *Corylus* also from certain parts of the States. This is quite a limited list, though of course there are special regulations governing the importation of nursery stock. But even in the above list there would seem to be some anomalies. In the regulations against potatoes—directed largely against wart-disease—one notices that potatoes from Europe are refused admission altogether while potatoes from the wart-infested districts of Pennsylvania, West Virginia, and Maryland are allowed in if provided with a certificate that they were grown outside the infected and quarantined area and have been examined and found free from wart disease. A similar, and I am sure equally reliable, certificate could be given in England, and some explanation seems called for. I cannot help feeling that the differential treatment in a case like this is due to an old distrust of the phytopathological service in England, a feeling which, however justified in the past, has now ceased to have any basis in fact. One of the main advantages of conferences such as this is that the people responsible for framing and carrying out these regulations are brought into touch with one another and mutual confidence is engendered. Everything hinges on the development of confidence in the reliability of the inspection service in the exporting country and, of course, unless the service is kept at a high pitch of efficiency there can be no real confidence. I am quite sure that as long as it is possible for a country to send out heavily diseased consignments, no matter what the plant or what the disease, so long will it be impossible to get importing countries to relax their restrictions. There are signs that the importance of growing clean and healthy stock for propagation is more fully recognized than it used to be. The potato seed certification movement is a proof of that.

The position at present in the overseas parts of the British Empire is somewhat as follows.

[5] Dorsey, M. J. Symposium on crown gall inspection. Proc. Amer. Soc. Hort. Sci. **1923**: 255–256. 1924.

In many countries prohibitions are in force to the effect that no plants of particular species may be introduced from any part of the world or from certain areas, as the case may be. Such prohibitions are likely to remain, but the instance quoted in regard to the Canadian potato regulations shows that they require examination from time to time to see whether they make distinctions not justified by the facts.

In many countries all plants or certain large categories of plants, for instance all nursery stock, can only be imported under permit and accompanied by health certificates, being subject to re-inspection on arrival. No one can properly object to these requirements, provided the inspection on arrival is properly and reasonably carried out. Most of the trouble has arisen on this point. The inspectors are only human and if they find consignments coming in that should not have been sent they are liable to get stricter and stricter in regard to the country sending such consignments. It is necessary, therefore, to do everything possible in the exporting country to prevent diseased consignments being sent and unreasonable to expect any relaxation of the strictness of the inspection in the importing country until the exporting one has set its house in order.

It is, I think, quite possible that restrictive measures against the entry of living plants have reached their maximum severity, as far as concerns imports from England and other countries that have taken steps to improve the cleanliness of export consignments. I believe we may reasonably hope to see the severity of these measures gradually relaxed, as confidence in the British inspection service grows. In other words, one may be permitted to hope that, while every importing country will still prohibit certain things, the list of these will be a small one; and that, though powers will still be held to reject obviously diseased consignments, these will be severely applied only against certain major diseases not already present in the importing country while, as regards the rest, good stock will be admitted even if it includes an occasional parasite.

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SOME ASPECTS OF THE MORBID ANATOMY OF PLANTS

BY

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SOME ASPECTS OF THE MORBID ANATOMY OF PLANTS

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(With Plates VIII–XII and 13 Text-figures.)

ADDRESS OF THE RETIRING PRESIDENT OF THE ASSOCIATION OF ECONOMIC BIOLOGISTS, DELIVERED ON JANUARY 24TH, 1930.

Regeneration.

The work of Němec (24), Vöchting (43) and others has shown how completely the tissues of growing parts of plants, destroyed by traumatic action, can be, in numerous cases, regenerated from the most diverse sources. Not only can the conventionally distinct derivatives of the dermatogen and periblem be reconstituted from those of the plerome, and a cortex cell (if it be not too old) directly become epidermal by thickening its outer wall, acquiring a cuticle, and even growing out into a root hair, but the entire lost halves of a young tuber, stem, or petiole may be regenerated under suitable conditions in form and structure scarcely distinguishable from the normal.

Vöchting has also shown that a perforating wound may become surrounded, not only by a cork, but by a deeply seated cambium which may cut off phloem towards the wound and xylem deeper in, the part between the phloem and the cork coming to resemble a cortex.

That this process may be carried so far as the development of an epidermis lining the wound cavity would appear to be established by some experiments described by E. F. Smith (41). He inoculated young flower heads of sunflower with the crown gall organism, *Bacterium tumefaciens*, by needle stabs, and others were similarly treated but with a sterile needle. The result in both cases was the development of long cavities in the pith of the rapidly elongating stalk. That shown in Plate VIII, fig. 1, was 16 inches below the inoculated apex when cut, this distance representing elongation after inoculation. It shows the cavity surrounded by a more or less complete vascular ring with xylem outside, then a cambium cutting off phloem on the wound side, then bundles of fibres of the hard bast type, next a thin-walled parenchyma

resembling cortex, and finally, lining the cavity, an epidermis sometimes smooth, sometimes with hairs like those on the surface of the plant (Plate VIII, fig. 2). Quite similar structures, even to the epidermal hairs, were got when the sterile needle was used. Smith makes a suggestion that these structures may possibly result from carrying in, on the point of the needle, invaginated fragments of the meristem of the torus which become grafted or transplanted, inside out, in the pith and subsequently elongate with the growth of the plant. They are, however, more easily explained as regeneration phenomena like Vöchting's. Even naturally formed hollows in the stem of *Brassica oleracea* were found by Beijerinck(2) to become surrounded by a cambium cutting off phloem towards the wound and xylem on the outer side, and Smith(39) artificially induced a similar condition by injecting monobasic ammonium phosphate into the hollow of young internodes of *Ricinus*.

Wound repair.

The commonest form of replacement of lost tissues is by the process of callus formation, such as takes place in the healing of wounds under moist conditions.

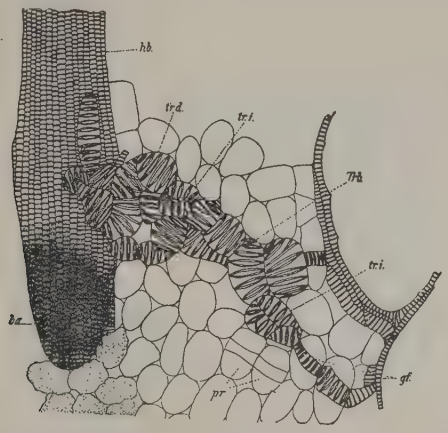
Callus may be formed from any living cell whatever under appro-

Text-fig. 1. Tracheid formation in the callus from a root of *Beta*. (After Küster.)

priate conditions, and the subsequent changes undergone in the callus mass are independent of the origin of the tissue.

The living cells bounding the wound that have not been killed in wounding grow out and form a tissue of dividing thin-walled parenchyma. In the deeper part of this the first differentiation frequently takes place

by the direct transformation of a callus cell, derived from any tissue, by reticulate thickening and lignification of its walls into a tracheid-like element (Text-fig. 1). This is one of the key processes in morbid anatomy. Several of these tracheids often develop close together, so that a little island of woody tissue results. Neighbouring groups may unite to form irregular tracheidal strands, varying in length and often broken; or an isolated group may become surrounded by a meristem developing in the cells bounding it on one or all sides, and this forms tracheids and wood fibres on its inner side. A procambial strand, forming wood and phloem, may appear at a somewhat later stage in the cells bordering



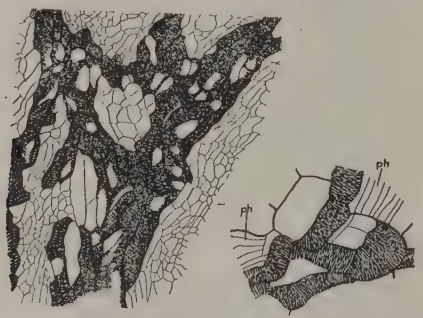
Text-fig. 2. Replacement of vascular connection destroyed by wounding in a leaf of *Impatiens*. Tracheids are formed directly from mesophyll cells (*tr.d.*) or after division of latter (*tr.i.*). A procambial strand is beginning to form at *pr*. (From Küster, after Freundlich.)

the tracheal strands, and may join up with vascular outgrowths from the original conducting system of the organ. Text-fig. 2 shows how readily resting cells of a tissue can, even without forming callus, restore a broken conducting tract by the direct or indirect transformation of the ground tissue into tracheids, outside which a new procambial strand may form. Sometimes, however, meristems develop in a callus not in association with tracheids.

Similar processes are extremely common in woody galls, the example shown in Text-fig. 3 being an island of tracheids in raspberry crown gall from a figure by Wulff (47). Phloem is not mentioned by him in these islands, but phloem strands are frequently found in the similar galls on

Rubus occidentalis that I have examined. The vascular system that supplies the adventitious cotyledonary leaves that are formed, as described by A. W. Hill(11), when the swollen hypocotyl tuber of *Cyclamen* is decapitated, arises, as was shown by Boodle(4), by division of one or more of the normal cortical cells (Text-fig. 3). There may be a central mass of xylem surrounded by phloem, or xylem and phloem pursue a wavy course, often rather widely separated from one another, towards the vascular ring. The elements in all these cases are very short, being limited by the diameter of the parenchyma cells from which they are formed, and the similarity of both processes is obvious.

The outer free zone of a callus may become corky by the development of a cork cambium, or may be a loosely united mass of cells re-



Text-fig. 3. On left, tracheid island in raspberry crown gall, with indications of meristematic activity in the surrounding gall parenchyma. (After Wulff.) On right, portion of trace of adventitious leaf of *Cyclamen*, cut longitudinally. *ph*, phloem. (After Boodle.)

sembling what is termed hyperhydric tissue. This arises from a meristem which cuts off roundish, loosely attached cells, resembling lenticel tissue, outside and thin-walled, callus-like cells inside. Similar tissue may form at the free surface without the intervention of a meristem. Very frequently also a large transverse cambium develops below the free surface of the callus and cuts off wood on its inner side and, less often, phloem and secondary cortical tissue on its outer side. Occasionally a layer resembling an epidermis clothes the surface. This is more frequently developed when a petiole or stem is split longitudinally, the callus outgrowth then, in favourable conditions, reconstituting the central cylinder by a cambium joining up with that of the uninjured half and forming not only phloem but a more or less normal cortex and epidermis outside.

The final stage of differentiation in a callus may be the exogenous formation of shoots from a mother cell or cell group at the surface of the callus or a few layers in from the surface. Even a callus derived from pith cells can give rise to these shoots. Their formation seems to be stimulated by excessive nutrition, for in uncongenial grafts (such as those between different species or genera of Solanaceae(16)) the upper side, which gets an excessive supply of nutriment from the scion, may form a callus very rich in shoots. Roots are often formed from callus, but, unlike the shoots, come always from the deeper layers. The various types of leafy outgrowths found at times on certain galls, such as the so-called embryomata of crown gall, are no doubt to be likened to callus shoots and need not be taken to imply (as has been suggested) that certain specific cells or cell groups preserve throughout their development a distinctive embryonic character, capable of regenerating the whole shoot system. In the processes leading to the formation and differentiation of callus, evidence can be found, in support of that given by other regenerative processes, that all living cells of the plant body have, at least, the potentiality of forming all parts of the plant. One of the effects of parasitism is, in some cases, to call out this potentiality, though usually in an irregular or unrhythmic fashion.

In callus proliferations the newly formed cells retain the power of division for, theoretically, indefinite periods, so that cells far removed from the wound surface continue to divide and form ever-increasing masses of thin-walled parenchyma. A second type of wound healing, however, is common in which the process resembles the normal growth in thickness by being the result of the division of cambial cells, the products of which, as usual, soon lose the aptitude to divide, becoming differentiated. The tissues thus formed are sometimes termed wound wood and wound cortex.

The largest masses of wound wood are formed from the original cambium stimulated into activity by injury to the bounding cells from parasitic or other causes such as frost. Cankers are due in large part to an excessive development of this tissue. In the case shown in Plate VIII, fig. 3, a tea canker which will be further considered below, the cambium was killed in places and repair by callus from the sides has occurred. Elsewhere it has only been stimulated, and wound wood has formed in direct continuation of the normal wood. Quite similar wound wood may be developed from cambiums formed in any part of a callus mass as already described. In this case only xylem may be formed, without phloem. Sometimes xylem is cut off on the outer side

of the cambium, sometimes on the inner, when the meristem develops, as is not unusual, around a tracheid group or a mass of sclerenchyma or a localised injury. In the first case there may be a small phloem group in the centre of the nodule, in the second either phloem tissue or parenchyma develops outside the cambium. Knobby isolated masses of wound wood thus formed are found in the secondary cortex of the apple and other trees and are well known in association with the phloem disease of *Hevea* rubber termed brown bast(5). Very frequently the wound wood is developed in vortex-like masses of fibres and vessels, forming knots of very characteristic appearance, probably as a result of pressure from unevenly developing meristems or uneven growth at the junction of normal and wound wood. Structures of the same kind are also found in wound bast and even, occasionally, in the tracheid groups that develop in leaf calluses. They are extremely common in woody galls, and beautiful examples can be seen in the woolly aphis gall and the olive knot disease.

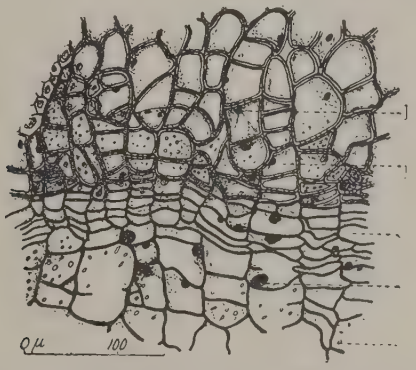
Histologically, wound wood differs from normal wood in having less regular rows, composed of shorter elements, in the usually greater proportion of wood parenchyma, which may be found in species in which the normal wood contains none, in fewer fibres or even their complete absence, in thinner vessels, and in larger medullary rays. Its less solid construction renders it liable to repeated damage. Thus the new wood in frost cankers is readily again injured by frost.

Wound cork.

The result of wounding may be the production neither of callus nor of wound wood but of wound cork. This is a common result in all organs of the plant, especially in relatively dry air. The case shown in Plate VIII, figs. 4 and 5, is from preparations by my colleague, Mr Wiltshire, showing a relatively early and a later stage of wound inoculations of apple twigs with *Nectria galligena*. The cork tends to form parallel to the surface of the wound and often differs from normal cork in having thinner walls and larger cells. Every living tissue from epidermis to cambium or pith may join in forming this cork layer, which always tends to unite at its margin with the surface covering of the sound part, epidermis or normal cork as the case may be.

In leaves its formation is sometimes accompanied by a certain amount of swelling due both to hypertrophy (generally taking the shape of an elongation of the cells towards the wound) and to hyperplasy, as the result of the formation of walls parallel to the surface of the latter.

In citrus scab, microtomed material of which has been kindly lent by Dr H. S. Cunningham, Plant Pathologist, Bermuda, the organism (which has received several names—*Cladosporium citri*, *Sporotrichum citri*, *Sphaceloma fawcettii*, *Sphacelia citri*) causes a necrosis of a few of the superficial cells of the leaf (Plate VIII, fig. 6) and provokes the active division of the spongy parenchyma (8). The new walls are roughly parallel, in the early stages, to the necrosed surface and the process extends to a considerable depth, sometimes leading, in attack on the under surface, to divisions in the palisade cells on the opposite side. Near the lesion the cells elongate more towards the injured part and become more divided than those farther away, and in the layer of long cells with several cross



Text-fig. 4. Edge of cavity in leaf of holly mined by *Phytomyza aquifolii*, showing elongation of the mesophyll cells and their division by cross walls to form a phellogen. Intercellular spaces absent. (After Kerling.)

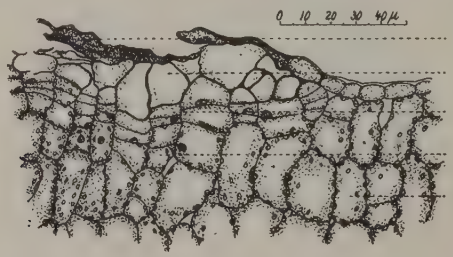
walls a cork cambium eventually develops (Plate VIII, fig. 7). This extends up to the epidermis all around the lesion and cuts off cork on its outer side and a little phelloderm on the side towards the sound tissues. All the primary cell walls of the hyperplastic area are thickened and intercellular spaces are much reduced.

Similar processes can result from insect injuries. Text-fig. 4 shows the margin of a larval passage of *Phytomyza aquifolii* (15) in a holly leaf, after the first year and after the insect has emerged. The passage has become more or less filled with large thin-walled bladder cells, and deeper in the mesophyll cells have elongated and formed a number of cross walls (up to 12), of which those towards the middle are close together and function as a phellogen, the cells on the wound side

becoming thin-walled cork, and those towards the healthy tissues phelloderm with thick, pitted walls.

Other types of injury can produce much the same effects. When savoy cabbage is exposed to tar vapour the leaves become spotted (15). The epidermis over the spots is killed (Text-fig. 5), while the first row of palisade cells elongates and may also broaden. The enlarged cells divide by three to five walls parallel to the necrotic surface, the outermost cells (those next below the epidermis) being very large, thin-walled, and suberised. The narrowest cells are about the middle of the original palisade cells and form a wavy line under the necrosed area, the cells on the outer side of this phellogen-like layer being corky and those on the inner side more normal and representing a phelloderm.

In most of these cases it is noticeable that the cork does not form directly under the wound, but is separated from the latter by one or



Text-fig. 5. Savoy cabbage leaf spotted from the action of tar vapour. Epidermis killed and a phellogen forming in the palisade cells. (After Kerling.)

more layers of large cells, sometimes bladder-like, and with walls and intercellular spaces impregnated with a substance resembling lignin but possibly a kind of wound gum. It is the cells below this zone that elongate and divide transversely, the middle divisions corresponding to a phellogen, with corky cells in rows outside and phelloderm rows below. There is usually a zone below the phelloderm in which the cells are enlarged, but little, if at all, divided. All this part is without intercellular spaces, and there is often no distinction between palisade and spongy parenchyma in it.

The metacutinised layers between the necrosed area and the cork correspond to the "blocking layer" of Priestley (30), and the whole process seems to agree closely with his description of the formation of wound cork.

All the living cells of the leaf may take part in these processes,

collenchyma and sometimes sclerenchyma cells (when young) apparently first losing their thickenings and then enlarging and even dividing.

When an organism penetrates the whole thickness of the leaf, the reaction is naturally restricted to the edges of the spot; the wound cork forms from the epidermis of one surface to that of the other, and the whole of the contained tissue may be cut off and fall out, producing the well-known shot-hole effect.

The cases cited show that there is nothing specific in the action of the stimulus. The reactions are similar to those found in the normal processes of the plant, *e.g.* below lenticels; they are latent in the plant; and the stimulus—fungus, insect, or injurious vapour—merely calls them into play. Even when the tissues from which it is derived are diverse, the tissue of reaction tends to be uniform. In the zone around the spots of *Clasterosporium carpophilum* on cherry leaves, for instance, not only is the differentiation between palisade and spongy parenchyma lost as a result of cell division, but the epidermal cells divide into cells very like the rest.

Lastly there are cases where no reaction follows the wounding, other than the deposit of suberin or lignin or wound gum in the walls of cells bounding the wound. There is no outgrowth of callus, no meristem to form wound wood or wound cork, and there may be no dividing walls formed in any of the cells near the wound. This type of healing is common in superficial wounds in roots and submerged aquatic stems, and Priestley (29) has correlated it with the presence of a well-developed endodermis, so that there is a barrier to the passage from the vessels of the solutes that are necessary for meristem formation.

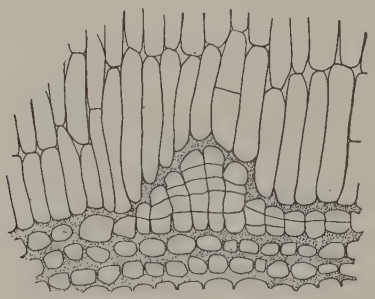
All sorts of intermediate conditions between these various reactions to wounding or necrosis may be found. Wound cork may form in part of a cut potato tuber and callus in another part. In one and the same cherry leaf spot caused by *Clasterosporium carpophilum* a good wound cork may be found in one place, while in another only the enlarged callus-like cells, becoming metacutinised, are formed. When an epidermis in some leaves is destroyed locally it may be replaced by a new epidermis with hairs and stomata derived from the mesophyll, or a kind of false epidermis merely with a cuticle but without hairs or stomata may form. This also occurs quite normally in citrus leaves, in which during development the upper epidermis often splits and is replaced from the mesophyll. There may be a very few cell divisions before cuticularisation. Here, as in many other cases, there is no sharp distinction between the regenerative and the callus processes of healing.

In general, every deep, closed wound or patch of necrosed tissue, even in secondary or so-called resting tissues, may become surrounded by a meristem, which may be directly formed from pre-existing cells, as in cork-cambium formation round the cavity, or may be preceded by cell proliferation to form a callus.

Anatomical changes caused by factors other than wounding.

All these processes of regeneration and repair result from wounding or injury. There is another great category of modifications in cell or tissue growth and differentiation arising from inner processes in which no gross injury is involved.

It is well known, for instance, that the mere placing of an actively growing shoot of many plants in unusually moist air causes the develop-



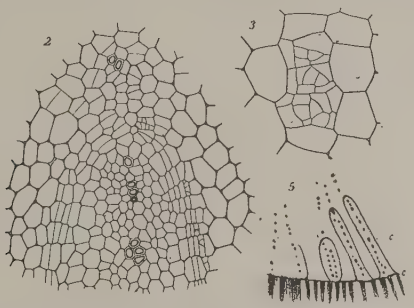
Text-fig. 6. Hypertrophy and hyperplasy of collenchyma of *Clerodendron* twig after vaselining. (From Küster after Schilling.)

ment of cellular outgrowths, intumescences, lenticular protrusions, and the like. These outgrowths are usually composed of rows of radially elongated, thin-walled parenchyma. In green stems and leaves the cells concerned are chiefly those of the epidermis, primary cortex, and mesophyll. These outgrowths can be produced even more readily if transpiration is checked by coating the parts with vaseline or paraffin. Text-fig. 6 shows how collenchyma (in the cortex of a *Clerodendron* twig) elongates and forms a thin-walled tissue after vaselining.

Not only humidity, but nutrition, light, and many other factors can cause deep-seated tissue changes. For such changes the terminology used by Küster (18) is convenient. When the result is under-development it is termed *hypoplasy*; when there is, on the contrary, a swelling, this may be due to a simple *hypertrophy* of the cells, that is to say, an increase

in size without division, or to a *hyperplasy*, which implies an actual increase in number of the cells. Sun and shade leaves, submerged or aerial leaves, etiolated shoots, starved or over-nourished plants all yield familiar examples of these anatomical modifications, due to what may be considered purely inner causes. How extensive such modifications may be was shown by some of Vöchting's (43) experiments.

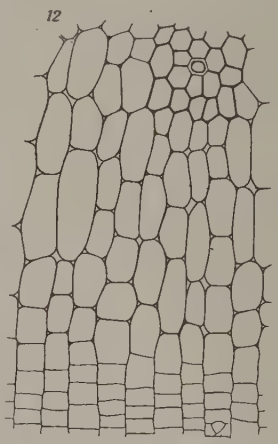
When the kohlrabi plant is prevented from flowering in the second year by removing the flower buds as they form, the stem above the tuber swells, the increase being very strongly marked in the leaf base cushions. The cortex and conducting tissues become much enlarged. The collenchyma divides and forms a thin-walled tissue. The phloem is radially



Text-fig. 7. Swollen leaf base cushion of kohlrabi. Lower right-hand figure, diagram of new cambium formations in the enlarged medullary rays, tending to encircle one or two rows of vessels, but leaving the innermost outside the ring. Left-hand figure, detail of inner part of a row of vessels with lateral cambiums uniting in the protoxylem region and a few phloem elements forming on the outer side. Cell division active around the innermost protoxylem vessels. Top right-hand figure, a phloem nest in the vascular region of a primary bundle. (After Vöchting.)

elongated but not otherwise much altered. The xylem of the main leaf trace may become split into a number of bundles, each more or less completely surrounded by its own cambium ring (Text-fig. 7). This is due to a new formation of cambium on each side of the enlarged medullary ray, the cells of which first elongate tangentially before dividing and the process either extending in from the original cambium of the leaf trace or beginning in the deeper parts and extending outwards. The ends of these lateral cambiums may unite, as the result of the radial elongation and division of a layer of wood parenchyma, in the primary xylem, which may be so situated as to isolate from the rest of the bundle some of the primary vessels on the inside. The new cambium forms

phloem outside and xylem inside. Sometimes the middle part of the protoxylem parenchyma may become very active and form a thin-walled tissue in which nests of phloem develop, which may then become surrounded by a cambium, on the outside of which an occasional xylem vessel may be formed. These small inverse concentric bundles are also formed in the broadened medullary rays, and the smallest ones may have no cambium or xylem.



Text-fig. 8. Thin-walled parenchyma formed on inner side of cambium in swollen axillary shoot of kohlrabi prevented from flowering. A primary vessel surrounded by thick-walled cells is seen near the top. (After Vöchting.)

When side shoots take part in a similarly induced thickening the swelling may be due largely to increased cambial activity, but instead of normal wood a thin-walled tissue is formed (Text-fig. 8), inside which some of the primary vessels, with thicker-walled tissue around them, may be seen. The phloem is much increased in this case also.

In similarly treated savoy cabbage Vöchting found concentric inverse bundles in the thickened roots and leaf base cushions, situated in the collenchymatous tissue of the inner xylem, though no bundles of this type are known in the normal plant as they are in normal kohlrabi. In the stem thickenings thus produced when the savoy is prevented from flowering, the phloem is much enlarged and, both in this plant and the kohlrabi, there is a considerable development of stone cells and of peculiar elongated branching sclerotic elements, termed idioblasts by Vöchting. These are found in the same region in some of the fungus galls to be referred to later. On the other hand, true fibres are noticeably scarce in the wood and phloem of the abnormal tissues in both cases.

One other experiment of Vöchting's requires mention. He succeeded in grafting young kohlrabi leaves into the medulla of a tuber, where they grew into abnormally large and thick organs. In the thickened petiole he found the bundles tended to become concentric.

Hans Winkler, a year earlier(45), had obtained a still more striking case of this formation of concentric bundles in the petiole. He was able to get well-developed flowering shoots from the leaves of *Torenia asiatica* cut off with the petiole and planted so that the latter rooted. The petiole

thickened when it thus came to function as a stem, and its normally flattened collateral leaf trace bundle became a round, closed woody cylinder (Plate IX, fig. 8). The change took place by the development of cambial divisions (Plate IX, fig. 9), at first in the old cambium which had become inactive and then in cells of the ground parenchyma, either extending out from the side of the old cambium and curving round till the two sides united, or in groups of isolated parenchyma cells opposite the central part of the leaf trace, the process spreading laterally and curving down to join the original bundle cambium. In the case shown, the former method has occurred and only the two cells in the middle above have not yet fully joined in the process of division. Some of the products of division have become rows of tracheids, and phloem elements are appearing in places. Ultimately there forms a compact woody cylinder at least twenty times the area in transverse section of the normal leaf trace. A small-celled phloem surrounds the wood, and all the elements formed outside the original bundle are short as they are limited by the diameter of the ground tissue cells.

Enough has been demonstrated in numerous investigations to establish the fact that, by purely internal processes and without the intervention of any parasite or agent of necrosis, every living tissue can become hypertrophied and all, by cell division, can show hyperplasy. In the hyperplastic tissues differentiation to other tissue forms can follow. Naturally, cells that have not finished growth and differentiation react the more readily, but even old cells can be affected, either directly or after first dividing and thereby becoming, so to speak, rejuvenated. Even cells that have developed thickened walls, as collenchyma, can enlarge and divide, or those in the early stages of lignification can be delignified, resume the parenchymatous condition, and behave like any other parenchyma. Differentiation in hyperplastic tissues may come from direct alteration of a cell—perhaps only one or two of a group formed from a single mother-cell—to a thick-walled type such as a sclereid, or to form a little island of phloem or a tracheid; or it may be indirect as the result of the development of a meristem in the new tissue.

So also tissues, cells, cell walls, and cell contents can show hypoplasy, or arrest of development or of differentiation, from internal, more or less natural, causes such as unsuitable nutrition or a deficiency in light.

Comparison between gall formation and other anatomical modifications.

The processes that take place in gall formation, especially those due to vegetable parasites, resemble those described above, allowance being made for the fact that the stimulus may be more localised and may act for a longer time than in the case of, say, a wound. It seems very doubtful whether there is any essential difference between the tissue changes in gall formation and those in restitution and repair, or when tissues are stimulated or under-developed as a result of internal adjustments to alterations in the food supply or the environment.

That it may not be superfluous to reaffirm this point of view is apparent from a cursory survey of some recent phytopathological literature. Statements can easily be found that all tumours arise from cambial activity, or at most from certain embryonic cambial derivatives that have conserved their meristematic characters; that leafy shoots can only come from certain totipotent cells that have preserved throughout their developmental history the power of reconstituting the plant; that vascular tissue, when it appears in a gall at a distance from the main conductive system, comes from special groups of cambium derivatives that would normally have formed vessels but are deflected by some agency into forming parenchyma without, however, losing their vessel-forming character; that concentric vascular nodules or cylinders in the pith and cortex come from complex invaginations or evaginations of the cambium; and that the presence of such structures in a petiole or leaf implies an intrusive growth of stem-forming tissues into the leaf. It is obvious that some of these statements cannot possibly be correct. No theory of persistent initials will serve to explain the large transverse cambium often formed from all the cells in a layer near the surface of a callus, derived as they often are from the most varied tissues. Nor will such theories explain the regeneration of the *Cyclamen* cotyledons nor the production of leafy shoots on callus from all kinds of tissue.

Crown gall.

Many of these views have been based on the phenomena observed in crown gall caused by *Bacterium tumefaciens*. No other plant gall has been so completely studied, not only on account of its intrinsic interest but also because of its suggested analogy to cancerous growths in animals. In none can the anatomical changes be better followed on

account of the magnificent series of photomicrographs with descriptive text in the numerous publications of Erwin F. Smith.

When growing stems of sunflower, tobacco, tomato, castor, etc., are inoculated with the crown gall organism by means of needle pricks, it is not uncommon subsequently to find closed vascular rings, spheres, or cylinders in the pith and cortex(42). When found in the pith the orientation of the tissues is frequently inverted, the phloem being inside and a ring of xylem outside the cambium. In the cortex and petiole (Plate IX, fig. 10) the orientation is normal and, as already mentioned, Smith interpreted these structures when found in leaves as evidence of an actual infiltration of cells bearing stem characters by a process similar to the infiltration or metastatic processes in animal cancers. The examination of his photomicrographs of the earlier stages of this formation, however, shows that it comes about as a result of division of the ground cells of the petiole outside the leaf trace. In one of these (Plate IX, fig. 11) the new cambium has as yet only joined up with the original cambium on one side. In another, at a later stage, the gap has been almost entirely closed (Plate IX, fig. 12), but there are still some ground tissue cells in the early stages of division. The centre of the new formation is a mass of tumour cells, but it is sometimes possible to make out that cells of the ground parenchyma are included within the ring. Robinson and Walkden pointed out in 1923(36) the resemblance of this process to that described by Winkler in *Torenia*, and showed (what is already evident from Smith's photographs) that it resulted from the division of ground tissue cells opposite to the protoxylem, the latter being the region that they proved was occupied by the bacteria. Riker also(35) independently observed, in the same year, a tumour strand in the outer cortex of inoculated sweet-pea giving rise to a secondary gall with a closed vascular sphere (Plate IX, fig. 13). Serial sections showed that there was no connection between this vascular nodule and the vascular cylinder of the stem. Like Robinson and Walkden, he found (and this has since been confirmed by J. B. Hill(12)) that tumour-strand formation in this position and in the pith is due to extension of the bacteria along intercellular spaces running longitudinally in the stem (Plate IX, fig. 14) or drawn out by rapid elongation of young parts, the cells bordering the infected spaces being stimulated to rapid division. In the pith strands formed in the sunflower Riker found some showing only hypertrophy of the cells round the first-formed tumour cells, but in others the vascular ring was developing, with xylem outside.

Magrou in 1928(21) endeavoured to explain the closed vascular rings

that he also observed in his crown gall inoculations by a hypothesis that is more ingenious than convincing. He supposes that the cambium is stimulated to tangential hyperplasy and, having no room to expand laterally, becomes puckered into folds, some of which bend into the pith and others out into the cortex, in both cases tending ultimately to become cut off from the parent cambium. This would explain the inverse orientation in the pith and normal arrangement in the cortex that has usually been observed. Or he supposes that in needle-prick inoculations the ruptured cambium grows out and in along the edges of the wound, uniting at its base in the pith but recurving in the cortex where the wound prevents union. This explanation altogether fails to account for the concentric leaf trace bundles shown above, or for the absence of any remnants of a vascular connection with the main conductive system, or for the fact that the centre of these structures is usually occupied by a core of tumour cells, often in longitudinal connection, above and below, with a non-vascularised strand of such cells extending from the point of inoculation. Still less would it account for the similar structures shown by Smith in the wood ring itself, situated in the protoxylem region, where he has also figured a little nest of phloem in an inoculated sunflower. It will be remembered that Vöchting obtained similarly situated phloem nests in his kohlrabi plants prevented from flowering, and found that they represented an arrested early stage of inverse bundle formation.

There need be no doubt that Robinson and Walkden's and Riker's explanation of these formations as due to the presence of bacterial infection in elongated intercellular spaces or protoxylem vessels, acting as a stimulus which causes hyperplasy of surrounding cells, is correct, and that they are not associated with any activity of the normal cambium but are at a distance from, and quite independent of it.

The inverse arrangement of the bundles of the pith, when such are found in crown gall, need cause no surprise. Medullary bundles are not uncommon in many families of plants and are frequently concentrically inverse or sometimes composed of phloem only (13, 22). Various explanations, not excluding one on which Magrou's theories are based, have been offered of their inverse arrangement, none of which appears very convincing.

Normally orientated cortical bundles or nodules, isolated from the main conducting system of the stem, occur in at least two other woody galls due to bacteria. In the olive knot caused by *Bacterium savastanoi* I have found beautiful examples, and they occur also in the bacterial

tumours of the Aleppo pine described by Prillieux(31), Petri(27) and Dufrénoy(9), the latter author, who has figured one, taking it to represent the core of an adventitious bud which had failed to emerge from the cortex and remained permanently buried. Petri, however, showed that they were formed by a meristem developing around bacterial foci, as in crown gall.

Bacterial infections are perhaps more likely to cause tissue changes localised around a focus than are other parasitic attacks, as the organisms are commonly confined to a restricted space, such as a single elongated intercellular space. It is easy to find in even fairly old crown galls appearances such as that in Plate IX, fig. 14, where extension of the parasite along intercellular spaces in the subepidermal layers of a tomato stem at some distance from the point of inoculation has led to two small areas of commencing hyperplasy of the type that leads to the formation of "tumour strands." I have found quite similar structures in tomato galls 3 months old, kindly communicated by Mr R. V. Harris.

Though at first Smith believed these "tumour strands" to be actual intrusive growths of the tumour cells insinuating themselves between the pre-existing tissues, he later recognised that some at least resulted from the division of previously normal cells along the path of the strand and stated that "Possibly all do so"(40).

In the areas of rapid gall formation it is easy to follow the conversion of normal cortex cells into gall tissue. In Plate X, fig. 15, a tangential section has been cut of a tobacco stem gall. At *E.* the epidermis can be seen, at *Cr.* crushed cells in the outer cortex. Riker has shown(34) that the cells thus formed in tomato continue to be smaller and smaller by division up to over a month old, then enlarge somewhat. The average size of the normal cortex cell is nearly 5000 sq. μ, and this fell to 166 after 33 days, then rose again to 407 sq. μ in 58 days.

The tumour of the type shown breaks through the vascular cylinder by the path of the medullary rays. Fig. 16 is from a tangential section of the same tumour and shows the gall cells formed in a ray. During this process extension up and down—mainly up—may take place in the form of narrow strands, marked here and there by little masses of gall tissue. These strands are usually in the protoxylem in *Chrysanthemum frutescens*, a plant in which the apparent upward extension is due, as Robinson and Walkden have shown, to elongation of the infected part.

The crown galls hitherto mentioned have been in herbaceous stems, and in the early stages these are marked by localised areas of reaction in the cortex, vascular cylinder, or pith. Another type of considerable

interest is found in the more or less woody stem or cane of the raspberry. In a fruiting cane, that is a cane in the second year of growth, the galls appear on the stem of the American black raspberry, *Rubus occidentalis* (specimens of which were kindly communicated by Dr Riker), as multiple outgrowths on the above ground parts. A transverse section of one of these in the early stage (Plate X, fig. 17) shows that the hypertrophy is at first confined to the region of the pericycle outside the fibre bundles. There are no nest-like groups of tumour cells such as are found in the plants previously considered. The swelling consists of thin-walled radially elongated parenchyma in rows and with scanty contents. The multiplying cells lie below the cork and are at first external to the groups of pericyclic fibres. At this stage they may be considered to be a parenchymatous phelloderm, but the reaction soon extends down between the fibre bundles and involves the tissues between them and the outer part of the phloem. The growth in this position bends up the pericyclic fibres and separates them widely from one another. The hypertrophy and hyperplasy rapidly increase (Plate X, fig. 18), but there is little cambial stimulation. Here and there (usually not over the whole arc of cambium underlying the gall) there is a small outgrowth of gall wood, which is sharply differentiated from the normal xylem by the scarcity of fibres and the thinner walls and wider lumina of the vessels, which are often distorted in their course. Outside this a patch of active cambium is found, and over it the phloem. The margin of the tumour is still mainly pericyclic, but the central mass is of more doubtful origin, being probably partly pericycle, partly phloem and even possibly partly cambium. Out in the gall parenchyma there early appear single tracheids or tracheid groups, just as in an ordinary callus and, like these, extremely short. One of these groups from the gall on the allied *R. idaeus* is shown in Text-fig. 3 on p. 178. As in callus, too, the tracheid groups sometimes become bordered by a meristem, and develop further tracheids on one side and phloem or gall parenchyma on the other. The gall often becomes highly vascularised by this means and is penetrated in all directions by irregular vascular strands, some of which can be traced into the original vascular cylinder.

At the surface the cork is early ruptured, and though there may be some attempts at renewed cork formation, the surface of the gall is usually a loose mass of brown collapsed cells giving a weak lignified membrane reaction, and resembling the so-called hyperhydric tissue already mentioned as one of the tissues frequently formed on the surface of a callus.

Sections cut from galls resulting from inoculation with *Bacterium tumefaciens* on the ordinary red raspberry, *Rubus idaeus*, kindly provided by Mr R. V. Harris, resemble those on the other species, though the well-marked corky layers that form the so-called polyderm in the endodermal region make it even easier to determine the origin of the gall below this position.

Olive knot.

In the olive knot caused by *Bacterium savastanoi* (1, 31) the galls are also formed on woody stems. In the primary gall the part external to the cambium is mainly involved, the cells of the outer cortex, immediately below the periderm, forming an important part of the gall, which may, however, involve the inner cortex, the pericycle, and the phloem, all of which may show a marked hyperplasy around the bacterial pockets. The latter are more marked than in crown gall, as the organism causes necrosis of small masses of tissue so as to form obvious cavities. The swelling pushes out and ruptures the cork (a subepidermal one in the olive), and also separates the bundles of pericyclic fibres from one another. The process extends to the phloem parenchyma, so that the sieve tube groups and fibres are similarly dispersed. It becomes almost impossible to differentiate the various tissues external to the cambium from one another, phelloderm, cortex, pericycle, and phloem being composed mainly of oval gall parenchyma interspersed early with pitted sclerenchyma and tracheids. The xylem is not directly involved, but the cambium is stimulated into increased activity to a certain extent so that the woody cylinder becomes thicker than normal, while the medullary rays are increased in diameter. The bacteria reach the inner wood probably along the rays, and extension, mainly in an upward direction, occurs in or near the protoxylem, where the tissues seem to be particularly readily penetrated by pathogenic bacteria. This migration of the bacteria results in the formation of secondary tumours as in crown gall. In these secondary tumours Schiff-Giorgini (37) states that the bacteria are always found in the wood.

In the cortical gall tissue of olive knot, tracheids eventually appear and meristems may form which develop spherical or cylindrical nodules of wood surrounded by a cambium, which may cut off phloem or parenchyma on the outer side. When the strands are elongated, the usual twisted or vortex type of tracheid tissue may result, as in callus wood. By the time the galls have reached a fair size they are largely composed of lignified tissue, and the bacterial colonies are found in the

midst of nests of gall wood, around which is still a fair amount of gall parenchyma. The vascularised tissue of the gall unites with an outgrowth of traumatic wood from the vascular cylinder of the axis, which spreads out into the gall.

In the form on the oleander(38), caused by a variety of the same organism, the infection channels are stated to run chiefly in the cortex and the cells around the bacterial cavities show hypertrophy and hyperplasy, resulting in a sheath of small, thin-walled, cuboidal cells.

Other gall formations in tissues mainly external to the cambium.

Another type of gall in which there is no indication that the cambium is the tissue mainly concerned, due this time to an insect, is that caused on various conifers by *Dreyfusia nüsslini* so well described by Chrystal(6). Here again the stimulus is localised, even more so than in the bacterial galls, and probably, too, acts for a shorter period than in the latter. The attack (which may be multiple) takes place at an early stage in the bud or just below, in the former case sometimes before lignified xylem has been developed. It causes an arrest in the formation of xylem, and hypertrophy of the phloem and medullary rays, sometimes extending through the latter to the pith. The stylets can sometimes be traced half-way down a ray but usually end in the phloem; they are unable to penetrate the xylem after lignification sets in. In their immediate neighbourhood the cambium ceases activity. In early attacks, before xylem has been differentiated, the whole or a part of the procambial strand in the neighbourhood of the stylet becomes a gall tissue of large, densely filled cells with prominent nuclei. In attacks lower down the shoot, the cortex may also be involved.

Sometimes in multiple attacks the xylem is separated into isolated bundles, and when this occurs a cambium may form laterally along each bundle in the medullary rays and internally in the pith, so that the whole bundle becomes surrounded by cambium. The tendency for bundles isolated by enlargements of the medullary rays to become surrounded by cambium was already noted in Vöchting's experiments described above, and is one that is frequently observed in both insect and fungal galls.

In the gall formed by *Phylloxera vastatrix* on vine roots, attack on a young root may equally lead to an arrest of development, the xylem being replaced by parenchyma where the stylet has penetrated, to a greater or less depth—sometimes completely in very early attacks before

differentiation has taken place. The rest of the root cortex in this case hypertrophies and the cells divide freely, while the xylem has short and very broad vessels. Petri(28) has clearly shown, however, that in the *Phylloxera* gall the effect on the tissues depends on the age of the root attacked. In somewhat older roots, where differentiation has progressed further, the pericycle, which is the region of cork formation in vine roots, shows hyperplasy all round, and the cortex becomes swollen laterally. Still later the cambium reacts actively, cutting off parenchymatous xylem internally of the same type, as will be mentioned later in the woolly aphis gall and, like the latter, becoming more normal again as the stimulus passes off, while the now well-developed pericyclic phelloderm of the secondary cortex is also multiplied. In still later stages the reaction fails to reach the cambium, and the swelling is chiefly due to the parts external to the phloem.

These cases are enough to show how incorrect is Dufrénoy's statement(9) that tumefaction is essentially a cambial activity, or M. T. Cook's(7) that galls only result when the excitation or stimulant is applied to the meristematic tissue.

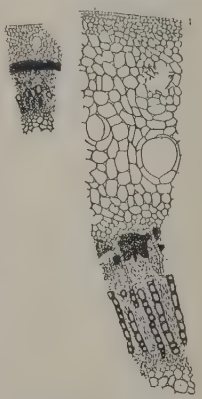
Galls involving cortex and vascular cylinder.

Witches' broom of cacao.

The next type of gall to be mentioned is one in which cambial activity plays a part more or less equal to that of other tissues. In the witches' broom of cacao, caused by *Marasmius perniciosus*, the stem is abruptly swollen to twice or three times the normal diameter. The swelling may involve everything from the epidermis to the centre of the stem. In a case measured by Went(44), the cortex, phloem, and xylem were each about three times their normal thickness, and the pith nearly twice as broad as lower down. The relative amount of thickening in the different tissues is, however, very variable. In the section shown in Plate X, fig. 19, the cortex is entirely crushed by the growth of the pericycle and deeper tissues. The intercellular hyphae of the parasite were found in the outer phloem near the point where the swelling commences, *i.e.* the oldest part of the gall. Here there is a slight proliferation radially of the one or two layers of pericycle just outside the outer fibrous layer and between the latter and one or two layers of inner cortex, rich in starch. Then there is a rapidly increasing proliferation of phloem parenchyma deeper in, which pushes apart the bundles of fibres in the phloem and separates them from one another. The crushed cortex

is still clothed by the remains of the piliferous epidermis. The brooms are short lived (some 6 weeks) and remain soft and fleshy, cork being rarely formed on them. In the cases described by Went, the mycelium at the base of the gall was confined to the cortex, and in his cases the cortex was not crushed but considerably thickened. Sometimes the xylem thickening may be about equal to phloem, pericycle, and cortex taken together. The medullary rays are broadened, especially in the outer part of the phloem, which is narrowed to a series of pointed wedges. It becomes almost impossible in this case to distinguish the cortical parenchyma from that deeper in, except that the pericycle can still be traced by its scattered groups of fibres. All the parenchyma, except that nearest the epidermis, comes to assume the same shape, elongated radially and with thin walls. This parenchyma multiplies by the division of all its cells, but mostly those of the inner cortex and outer phloem and, especially, the cells lying immediately next to the phloem fibres. The xylem vessels are broader and with thinner walls, and the outer xylem elements are largely parenchymatous. As one passes higher up the gall, the fungus is found throughout its thickness from the pith to the epidermis. The higher up one goes, too, the less clearly the different tissues are differentiated from one another. The fibres become at first septate, then reduced in numbers, and finally none forms.

Most of the thickening of the xylem seems to be from cambial activity, but in the other tissues it is mainly due to division of parenchyma *in situ*.



Text-fig. 9. Comparison of normal and swollen midribs of leaves from cacao, to show the effect of *Marasmius perniciosus*. (After Went.)

In the swollen leaves of this gall the distinction between palisade and spongy parenchyma disappears (as is common in leaf galls). In the midribs of sound and diseased leaves shown in Text-fig. 9, the cortex is over six times, the phloem nearly twice, and the xylem three times as thick in the latter as in the former. The xylem vessels are larger and more numerous, the medullary rays much wider and the pericyclic band of fibres broken up.

Acacia gall rust.

Another gall in which pericycle and phloem are extensively involved but cambial activity also occurs, is that of *Acacia decurrens* caused by the rust *Uromycladium notabile*, specimens of which have been kindly communicated by Dr G. H. Cunningham of New Zealand. In this case the relationship of the tissues is difficult to make out unless they are followed from the very early stages (Plate X, figs. 20 to 23). In the rachis of the bipinnate leaf there are two large bundle groups, each formed of a flattened band of xylem capped by a curved mass of phloem. The gall seems always to affect the bundle on the side opposite to that on which the leaflets are inserted in two rows near together. In the earliest stage (Plate X, fig. 20) there is a slight swelling in the middle of the phloem, where a group of cells becomes enlarged and thin-walled. This swelling extends, expanding the phloem into a conical mass (Plate X, fig. 21), the outer elements of which are large and thin-walled while those nearer the cambium are more normal. Soon the xylem is affected, and the new elements cut off from the cambium on that side show an excess of parenchyma with few wide vessels, though files of relatively thin-walled tracheids are still formed. There is an increase in the breadth of the rays and the formation of new ones and of much wood parenchyma. Fibres are still found scattered in the wood. The xylem hypertrophy, however, may not keep pace with that of the phloem and may come to an end fairly soon (Plate X, fig. 23). In the phloem there is a development of stone cells, and elongated fibre-like, thick-walled elements appear here and there, and seem to be similar to the previously mentioned idioblasts of Vöchting, which develop in the same region in Brassicas thickened as a result of the removal of the flower buds. The pericycle may also enlarge by the formation of radial rows of thin-walled cells, whose radial walls are in the same straight line. Groups of thin-walled dividing cells appear in the pericyclic fibre band and may divide the latter into segments. A cork may form in this zone, or the outer cells may proliferate as hyperhydric tissue. For a considerable time, however, the swelling may remain covered with the epidermis, bearing hairs.

In the early stages an irregular, sometimes discontinuous cambium can be made out between the xylem and phloem (Plate X, fig. 23), but the cambial cells usually lose their individuality later on and, with the outer part of the parenchymatous xylem, become involved in the general hyperplasy, so that it becomes impossible to differentiate between phloem, cambium, and outer xylem.

Differentiation of the type repeatedly referred to takes place by the direct change of some of the gall parenchyma cells into tracheids, or the development of isolated meristematic strands forming both xylem and phloem, which may be close together or diverge somewhat in their irregular course. Whorls and woody nodules are thus formed.

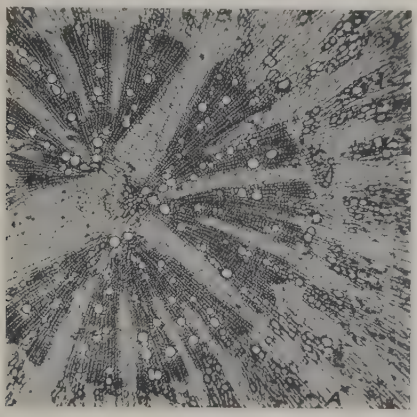
The hyphae in the early stage can be distinguished by their finger-shaped, sometimes branched haustoria in the enlarged phloem and, later, are easily made out in the pericycle and the vicinity of the cambium. Sections of the early stages have sometimes revealed them in the outer xylem, and in these cases the greater part of the young gall may be composed of modified wood. The galls, I am informed by Mr Rodda, Manager of the Te Kauwhata Horticultural Station, New Zealand, usually appear in late summer (February), and growth continues through the winter into November or December. The spore masses then appear. On small branches the galls usually die after the first crop of spores (insects often destroying them), but on large branches or stems they may go on growing for several years by renewed warty outgrowths, and ultimately reach a foot or more in diameter, being amongst the largest fungous galls known.

Galls involving mainly the vascular region.

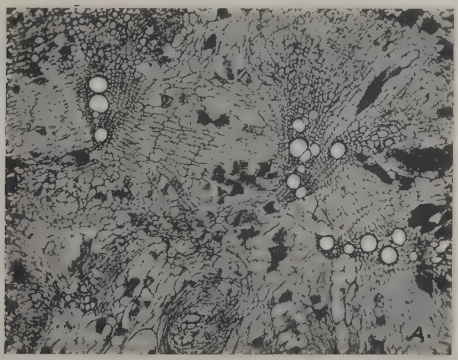
Club root.

There are a few galls that chiefly involve the vascular ring. In the club root of crucifers caused by *Plasmodiophora brassicae,* Kunkel(17) found that the swelling on cabbage stems caused by artificial inoculation at first affected the secondary cortex (the primary cortex having already been lost), but the organism reached the cambium fairly soon and produced a marked hyperplasy of the cells in this region. The elements cut off on the xylem side remained undifferentiated and again rapidly divided. In infections at later stages of growth, the medullary rays chiefly become infected and show a hypertrophy and hyperplasy which may be extremely marked (Text-fig. 10). Except at the point of original invasion, the phloem and secondary cortex only become infected outwards from the cambium. In stems which are no longer young, and in which the phloem tissues are hardened, only those cells that have developed after infection has reached the cambium become involved, if the medullary rays be excepted. The increase in size of the medullary rays may be such as to divide the vascular ring into widely separated

bundles. Distortion from this uncontrolled growth of the ray parenchyma may be such as to turn the bundles through 90 or even 180 degrees.



Text-fig. 10. Part of central cylinder of cabbage stem inoculated with *Plasmodiophora brassicae*, showing enlargement of medullary rays. (After Kunkel.)



Text-fig. 11. Later stage than Text-fig. 10, showing disorganisation of the wood by proliferation in the medullary rays. (After Kunkel.)

Not only is the original xylem broken up into separate masses (Text-fig. 11), but tracheidal tissue may develop in the hyperplastic ray parenchyma so that very confused structures result.

Woolly aphis.

In the tumours produced on the apple by the woolly aphis, *Eriosoma lanigerum* (32), the alteration is practically confined to the xylem ring of young shoots, the cortex, phloem, and pith being unaffected (Plate XI, fig. 24). In this case, it appears to be practically certain that the stimulus acts on the cambium mainly or exclusively. The first change is the suppression of the wood fibres, which are replaced by parenchyma; then the tracheids become thin-walled and are also largely replaced by parenchyma in rows of cells with pitted walls and sometimes containing starch. The newly formed wood is not lignified and mainly consists of wider cells than normal, with soft walls. Sometimes groups or layers of lignified wood are formed, especially in the later stages, so that patches of new xylem may appear at a distance from the old. Isolated cells or small groups in the deeper layers of the gall tissue may also become lignified. The medullary ray cells also hypertrophy and become thinner-walled than normal and, like the other parenchyma of the gall, divide in all directions so that their identity is lost. The sclerenchyma bundles of the pericycle become pushed apart by the radially elongated gall parenchyma, the unaltered cortex is split by the pressure, and the soft gall tissue protrudes. Cork forms on this, but in the winter cold the gall collapses and dies. Callus formation then ensues, new infestations occur on the callus masses and on the exposed gall tissue, and supplementary growths are produced.

It has been shown that the stylets reach the phloem, but do not penetrate further than to within three or four cell layers from the cambium, so that the stimulation of the latter acts at some distance. Why the action should be restricted to one side only, and that the side furthest from the stimulus, is not clear.

Tea canker.

Another case in which the gall is wholly woody is that known as tea canker, which has been attributed to various organisms, such as *Nectria* and *Macrophoma*, but the etiology of which is still obscure. The affected branches are swollen irregularly, the swelling usually surrounding the whole stem but being sometimes unilateral (Plate VIII, fig. 3), and the cortex is cracked and shows protrusions of reddish woody tissue. In the early stages of a thickening of the whole circumference, a transverse section (Plate XI, fig. 25) shows what appears rather like two annual rings of wood. Above and below the swelling, however,

these rings are not visible, but only a continuous body of xylem corresponding to the inner one. Hence the outer ring is a local formation, and as the other tissues are not appreciably swollen, the visible thickening is due to this excessive xylem development.

The new growth is due to cambial stimulation mainly on the xylem side, as in the woolly aphis gall. The gall wood elements are often not in radial continuation with the old, but are variously bent or contorted, sometimes even coming to lie across the rows. The pressure ruptures the cortex, and the bark becomes rapidly exfoliated into shreds. Sometimes the cambium is killed over a part of the circumference, and this leads to a complication of the structure by attempts at a normal process of wound repair extending in from the cambium at the sides (Plate VIII, fig. 3). The wound may be entirely closed, or repair may fail to close it and a gaping canker down to the original wood remains, bounded by swollen callus lips.

Another point of resemblance to the woolly aphis gall, and also to some of the *Phylloxera* galls, is that in many cases the more recently formed wood gradually comes to resemble the normal wood, with clearly differentiated vessels and fibres, as if the stimulus were passing off.

Sorosphaera veronicae.

Before leaving the consideration of galls involving the vascular system, a tumour that is usually confined to the procambial region behind the apical meristem may be referred to. This is the gall of *Veronica Chamaedrys* caused by *Sorosphaera veronicae*(3), one of the Plasmodiophorales and therefore in the same group as the club root organism. Specimens and microtomed sections of this have been kindly provided by Dr Schwartz.

In the longitudinal section shown (Plate XI, fig. 26) the tumour extends backwards on both sides from the point in the procambium where the xylem is just beginning to form, and also out into a leaf. Spiral vessels can be found throughout its length, but do not occupy any fixed position in the mass of gall tissue, being sometimes on its inside near the pith, sometimes on its outside towards the cortex and with only a few procambial elements between, sometimes in the middle. Evidently it is not only cells of the cambium, but also the derivatives of these lying around the protoxylem vessels, that become stimulated to increased growth and division. There is a great hyperplasy of the whole of the procambial region to form a tissue of more or less polygonal cells, in which are mingled the few xylem elements already differentiated

before the parasite reaches them and some scattered groups of elongated cells. Many of the infected cells and some of the neighbouring non-infected ones are also hypertrophied, sometimes greatly. As shown in Plate XI, fig. 27, there is no extension into the cortex or pith, but other sections have revealed a commencement of cortical invasion by an outward passage of the parasite from the vascular region through the endodermis. This invasion of the cortex from within, and the predilection of the parasite for the cambial region, are points of similarity to club root.

Galls involving primarily the pith.

Hypertrophies of the pith are commoner in insect than in fungous galls, as there is a large number of insects whose larvae inhabit the pith: many lepidopterous stem galls are of this type. The pith cells have remarkable powers of reaction by enlargement and division, and naturally when the woody cylinder is already formed in the stem the result of a pith swelling is that the bundles are forced apart and undergo various modifications.

In the insect galls due to larvae which occupy a pith cavity, the cavity is frequently surrounded by a nutrient tissue of densely filled cells formed by the division of the pith cells, and outside these is a zone of sclerenchyma, similarly derived, forming a protective layer. When the enlargement is considerable, these tissues are irrigated by vascular tissue joining up with the normal vessels of the stem. The stem bundles are forced apart by the enlargement of the medullary rays, and their number may be increased.

The example shown (Text-fig. 12) of the stem gall of *Potentilla reptans* caused by *Xestophanes potentillae*, Villers, from Houard's remarkable work on stem galls (14), indicates how several of these modifications come about. The cells around the gall cavity are elongated and much divided as a preliminary to the development of the nutrient tissue. Up to 30 or 40 cells may be formed by subsequent division in all directions in an original pith cell; in some other similar galls considerably greater numbers may develop. This hyperplasy extends into the medullary rays, and at the same time there is increased activity of the cambium, which forms secondary non-lignified xylem and parenchymatous secondary phloem. The cambial activity extends out into the greatly enlarged rays and down along the margins of the bundle, and may bend in at the perimedullary region so as to form a ring, or may extend towards the nutrient tissue of the gall. The irrigation of the latter is provided by

vessels developed from this cambium. The protective zone of sclerotic tissue appears between the new vascular tissue and the nutrient zone. In the formation of these protective layers a single mother-cell may contain parenchymatous daughter-cells in one part and sclerenchyma in another, a phenomenon frequently observed by Vöchting in his regeneration experiments.

The similarity of the process of development of these lateral cambiums, and of the parenchyma in the protoxylem region, to what has already been seen in the thickening due to prevention of flowering in the kohlrabi is evident. As already mentioned in considering the *Dreyfusia* gall, there seems to be a general tendency, when the medullary rays exceed a certain size, for the bundles to surround themselves with a ring of cambium.

In some similar galls, *e.g.* that produced by *Aulax* (*Aulacidea*) *hieracii* on *Hieracium umbellatum*, Houard notes that the newly formed small bundles may consist solely of phloem in the early stages, and when xylem appears it may develop around a core of phloem to form an inverse bundle, again as in Vöchting's experiments.

Text-fig. 12. Part of vascular region and pith of *Potentilla reptans* stem inhabited by the larva of *Xestophanes potentillae*, showing cambial activity extending down into the pith. The pith and ray cells are elongated radially to the bundle and much divided. The insect cavity is beyond the top of the figure, and the primary phloem at the bottom. (After Houard.)

Naturally the outer layers, pericycle, cortex, and epidermis, have to be modified to allow for the expansion of the stem that results from pith-inhabiting larvae. In some cases, *e.g. Epilobium montanum* stems swollen by the larvae of the lepidopterous *Mompa decorella*, an active pericyclic cambium develops, but instead of cutting off cork outside and phelloderm inside in the normal way, it forms thin-walled parenchyma on both sides. In this gall also there is an active formation of small cells in the hypertrophied endodermis, and also a very active division of the parenchyma cells of the internal phloem that is found in this plant. The cortical swelling may be due to simple hypertrophy, cells from a half to over one millimeter in length being found in the cortex in several of the galls described by Houard.

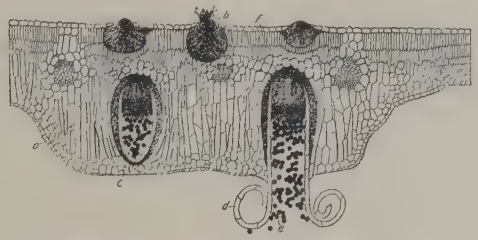
Galls restricted to the true cortex.

Purely cortical swellings, in which the central cylinder plays no part, are familiar in endotrophic mycorrhiza, where the endodermis of the infected root is not penetrated, and only cells of more or less defined cortical regions are hypertrophied. Some of the bacterial root nodules of the Leguminosae are also purely cortical formations, *e.g.* those of the French bean(23), though in others the central cylinder participates by a swelling corresponding in origin and position to a lateral root and joining up with the cortical swelling outside. In the common stem gall produced by *Protomyces macrosporus* on *Aegopodium podagrariae* there is a hyperplasy of the outer cortical cells restricted to those in the vicinity of the intercellular hyphae, the presence of which, in the early stages, can be postulated whenever a fully formed cortical cell is observed to be dividing. Though in this species of *Protomyces*, the vascular tissue is wholly unconcerned, in *P. pachydermus* on dandelion the spores occur in the phloem of the midrib and lateral bundles (Plate XI, fig. 28). This is only one of many cases in which two allied fungi produce different effects on the host, even when, as Magnus pointed out(19), both attack the same plant. Thus the aecidial stages of two species of *Puccinia*, *P. graminis* and *P. arrhenatheri*, both on the common barberry, produce, in the one case, localised leaf hypertrophies and in the other, witches' brooms, the differences being due, no doubt, to the widely different modes of infection in the two cases.

Leaf galls caused by fungi.

Leaves offer many excellent examples of the development, without the intervention of a meristem, of gall tissues composed of uniform cells, even when derived from cells that are normally quite distinct, as the palisade and spongy mesophyll. In the common abnormal growth caused by *Cystopus candidus* on the Shepherd's Purse (*Capsella bursa-pastoris*) the mesophyll of the thickened leaf is composed wholly of large round cells. Leaves of witches' brooms formed by species of *Taphrina* are also usually composed of round cells, with no differentiation into palisade and spongy layers and with small intercellular spaces. *T. deformans* also thickens the leaves, and causes the palisade cells to multiply and lose their normal shape, becoming almost isodiametric in the outer layer, where they are hard to distinguish from epidermal cells, and oval deeper in. *Exobasidium oxycocci* makes all the mesophyll into a uniform type of parenchyma without intercellular spaces in *Vaccinium macro-*

carpon and *Gaylussacia dumosa* (26). In *E. vaccinii* (46), on the other hand, the swollen leaves of *V. vitis idaea* preserve the palisade but little altered, the mass of the swelling being due to hypertrophy and hyperplasy of the spongy mesophyll, the cells of which are roundish or polygonal without intercellular spaces. In *E. rhododendri* on *Rubus ferrugineum* and *R. hirsutum* the effect is the same as in *E. oxycocci* when the leaf is totally infected from an early stage, but is like *E. vaccinii* when only a part of the leaf shows the swelling (10).



Text-fig. 13. Aecidial stage of *Gymnosporangium juniperi-virginianae* on apple leaf. (After Reed and Crabill.)

In the aecidial stage of *Gymnosporangium juniperi-virginianae* on the apple leaf (33), the hypertrophy is almost all due to the spongy mesophyll, the palisade being little altered (Text-fig. 13). The middle layers of the mesophyll are enormously elongated, and divided by transverse or oblique walls. It is in this tissue that the aecidia form, and its elongation gives room for the development of these organs in the deeper part of the leaf. Intercellular spaces are lost. This type of swelling resembles that sometimes found in leaves exposed to excessive moisture so as to cause the production of the intumescences already referred to.

Epidermal galls.

The epidermis may be directly involved in gall formation, as in the well-known host cells of *Synchytrium*, or may be indirectly concerned, as when it shares in the reaction of tissues around an invaded cell or other centre of irritation. In the latter case, it frequently tends to lose its characteristics as a distinct tissue. In the willow leaf galls caused by hymenoptera of the genus *Pontania*, Werner Magnus (20) found the epidermal cells dividing to form up to four daughter cells, and the underlying palisade up to eight, with a marked reduction in the differentiation between the two tissues.

In the capsule or sheath of small cells that forms around the single enlarged epidermal cell of the dandelion infected with *Synchytrium taraxaci*, microtomed material of which has been kindly provided by Dr W. R. Ivimey Cook, both epidermis and subepidermal cells are represented (Plate XII, fig. 29), though it is not possible to detect any difference in the daughter-cells of the two tissues. Here there is no cork formation, the small size of the source of the stimulus (localised to a single cell) and its relatively short duration, perhaps, being insufficient to induce any attempt to occlude the affected part. The cells elongate tangentially to the host cell and divide by walls at right angles to the latter. Intercellular spaces do not occur in this new tissue.

Localised stimuli that do not lead to cork formation are caused by several other epidermal parasites amongst the fungi. *Urophlyctis kriegeriana*, for instance, occupies a single epidermal cell, like *Synchytrium taraxaci*, but causes a somewhat more extensive reaction of the epidermis of the host (*Carum caruii*) with less penetration into the tissues below. On young potato stems the wart fungus, *Synchytrium endobioticum*, similarly stimulates the epidermal cells next to that in which the presorus develops to grow out into a rosette of club-shaped divided cells, and it is not until a later stage, when the resting form of the fungus has been carried deep into the tissues by repeated transverse divisions of the infected cells, and there are usually multiple infections, that the large warty outgrowths take place.

When *Taphrina aurea* penetrates the poplar leaf, the mycelium remains confined between the cuticle and epidermis, only sending short branches down between the cells of the latter. These are stimulated locally (Plate XII, fig. 30) and become two or three times the normal length, often dividing when they reach full size. There is little disturbance of the rest of the tissues.

Gall reaction to systemic infection.

At the extreme other pole from these cases of strictly localised parasites stand the diseases of the virus group. In many of these the morbid principle appears to be distributed throughout the whole plant. Few produce tissue reactions of the type discussed in this paper, but some do, *e.g.* the curly top of beet and Fiji disease of sugar-cane. In the woody disease of the passion fruit (*Passiflora edulis*), material and sections of which have been kindly provided by Dr R. J. Noble, who has studied it in Australia and proved it to be juice infectious(25), one of the main symptoms is a thickening of the pericarp, which may become

double the normal breadth and show irregular protrusions. The pericarp is abnormally hard and woody, due to extensive thickening and lignification of the cells. In the normal fruit (Plate XII, fig. 31) there is a single epidermal layer, then a hypoderm of three layers of thin-walled cells, below which is a band of lignified sclerotic cells also about three layers deep, followed by the ordinary thin-walled parenchyma of the main mass of the fruit wall. In the woody fruit (Plate XII, fig. 32) the sclerotic band becomes broader and more irregular, while the cells of the underlying parenchyma are also transformed in great part into lignified stone cells. At a later stage (Plate XII, fig. 33) cork forms below the sclerotic band, the outer layers of which (together with the epidermis) may become exfoliated. The phellogen develops in elongated cells, which divide as in the wound cork formation in leaf spots described above, but the cells on the side furthest from the surface become transformed into a sclerotic phelloderm, which shades off into the sclerotic tissues below. The local hypertrophies are due to this sclerotic phelloderm. In the well-known stony condition, termed lithiasis, in pears a similar sclerotic phelloderm is often formed, and several other cases of the kind will be readily recalled (one in the papaw is very familiar to residents in the tropics), but, so far as I know, this is the first that has been ascertained to be the result of a disease of the virus group.

That fungi and insects or mites can produce new structures in plants is undoubted, if by "new" we mean such as are not normally found in the life of the plant. The erineum hairs resulting from mite infestation, or the rosette cells of the epidermis around a *Synchytrium endobioticum* infection, are not like anything found elsewhere in the plant, but have a quite distinctive form. It is noticeable, however, that most of these cases occur in cells which, through the action of the stimulus, have become to a greater extent isolated from the neighbouring tissues than usual. Complicated correlation factors are probably involved, in which the stimulus of the foreign organism is, perhaps, very indirectly concerned. In the great majority of cases there is nothing new in the tissues of reaction, but merely a disorganisation or an intensification or inhibition of the normal processes of tissue formation and differentiation. The insect or fungus can do no more than call out powers which the cells and tissues already possess, powers which plants can ordinarily make use of in meeting such incidents of their life as may necessitate the healing of an injury, or the making provision for coping with unsatisfactory or unduly enhanced supplies of nutriment or light, or other

environmental vicissitudes. Not only the meristems have the ability to respond by the development of new tissues or by modifications in the products of their activity: other living tissues can play a part not less important. It may be that there are some tissues that react more readily than others: the cambium certainly does so, and perhaps the pericycle: but the examples cited above are sufficient to show that these differences must not be exaggerated. As Küster says: "In all living plant cells, even in the cells of the higher plants, there slumbers the potentiality for the development of all histological characters that appertain to the particular species." Many unnecessary complications of interpretation and perhaps some false analogies, in respect of the processes of morbid anatomy, can be traced to an insufficient recognition of this truth.

Besides the acknowledgments already made in the text to those who have been kind enough to communicate specimens and microscopic preparations, thanks are due to Mr L. A. Boodle, of the Royal Botanic Gardens, Kew, for much helpful criticism and advice.

Summary.

The anatomical modifications in the tissues of plants caused by the action of gall-inducing fungi and insects are illustrated by a number of examples, and compared with those produced by various processes of regeneration and wound healing or by factors that lead to anatomical changes without the intervention of wounds, such as nutrition, humidity, and the like. It is concluded that all living cells have the potentiality to react to various stimuli by hypertrophy, hyperplasy, or the development of meristematic tissues, and that the pre-existing meristems are not, of necessity, primarily implicated in gall formation.

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EXPLANATION OF PLATES VIII—XII

PLATE VIII.

Fig. 1. Medullary cavity in sunflower stem inoculated with crown gall by a needle wound in the young flower head, showing vascular ring, cortex, and epidermis formed around the wound. At *x* glandular hairs; at *t* an incipient tumour. (After E. F. Smith.)

Fig. 2. Detail of wall of cavity in Fig. 1, showing cortical tissue, epidermis and hairs. (After E. F. Smith.)

Fig. 3. Callus and wound wood formation in tea canker.

Fig. 4. Commencement of wound cork formation in apple twig inoculated with *Nectria galligena* 11 days previously. The cells outside the brown zone in the cortex and below the pericyclic fibres are elongating and dividing.

Fig. 5. Later stage of wound cork formation in apple twig inoculated with *Nectria galligena* 37 days previously. The wound is completely occluded.

Fig. 6. Citrus scab on rough lemon (*C. limonia*). Early stage showing elongation and division of mesophyll cells.

Fig. 7. Later stage of same, showing cork barrier completely cutting off the necrosed tissue. The original walls of the mesophyll cells are thickened and intercellular spaces almost absent. (Figs. 6 and 7 from sections by H. S. Cunningham.)

PLATE IX.

Fig. 8. Woody cylinder in rooted petiole of *Torenia asiatica* leaf bearing a shoot.

Fig. 9. Earlier stage of formation of same, showing renewed cambial activity in the leaf trace and new cambium forming in the ground-tissue cells on each side, curving round to unite at the top where two cells have only just begun division. The new cambium has formed rows of tracheids inside and phloem elements outside. (Figs. 8 and 9 after Winkler.)

Fig. 10. Petiole of *Chrysanthemum frutescens* about 11 weeks after an inoculation lower down in the stem with crown gall, showing leaf trace transformed into a closed woody ring, in the centre of which is a group of tumour cells in continuity with a strand of similar cells in the protoxylem region down to the point of inoculation.

Fig. 11. Early stage of similar leaf trace, showing new cambium forming on the right-hand side in the ground-tissue cells.

Fig. 12. Later stage from same petiole as Fig. 11, but lower down, showing new woody ring almost complete. (Figs. 10 to 12 after E. F. Smith.)

Fig. 13. Secondary crown-gall formation in sweet-pea stem inoculated lower down about 7 weeks previously, showing closed woody nodule in the cortex.

Fig. 14. Two subepidermal intercellular spaces infected with *Bact. tumefaciens*, showing hyperplasy of surrounding cells to form a more or less concentric sheath. (Figs. 13 and 14 after Riker.)

PLATE X.

Fig. 15. Tangential section of tobacco stem crown gall, showing hyperplasy of cells of cortex to form tumour tissues. At *Cr.* crushed cells; *E.* epidermis.

Fig. 16. Tangential section of same tumour as last deeper in, showing tumour tissue in wood and commencement of extension vertically. *Tr.* tracheids. (Figs. 15 and 16 after E. F. Smith.)

Fig. 17. Early stage of crown gall of black raspberry (*Rubus occidentalis*), showing hypertrophy and hyperplasy of cell layers between cork and pericyclic fibres. Vascular region unaltered. On the left the cortex has begun to split above the phellogen. Note absence of tumour tissue nests.

Fig. 18. Later stage of same, with all the tissues down to the cambium involved. Outer part of xylem broadened below tumour by increase in the medullary rays, and in this part there are no large vessels. Note the bending up of the pericyclic fibres on the right, from a proliferation of the phloem tissues.

Fig. 19. Longitudinal section of swollen cacao shoot from the witches' broom caused by *Marasmius perniciosus*. The primary cortex of the normal part (on the right) has been bent up and crushed by a pericyclic proliferation in the thickened part. Hyphae were found in the pericyclic region of the thickened part near its point of origin. Further to the left the phloem is also much enlarged.

Figs. 20 to 23. Progressive early stages of gall formation on *Acacia decurrens* by the rust *Uromycladium notabile*. In Figs. 21 and 22 the thin-walled phloem hypertrophy is visible, in Fig. 23 the wavy dark line about the centre of the gall represents the position of the cambium, but there is little difference in the cells on each side of this.

PLATE XI.

Fig. 24. Woolly aphis gall on apple twig, showing parenchymatous xylem becoming normal again at the periphery. Tissues outside the cambium not affected. (From a section cut by S. P. Wiltshire.)

Fig. 25. Tea canker, showing wound wood simulating an annual ring. Note its looser structure and scarcity of fibres, except at the periphery where it becomes more normal.

Fig. 26. Longitudinal section of *Veronica chamaedrys* affected by *Sorosphaera veronicae*, showing gall formation in the procambial region and extending out into a leaf.

Fig. 27. Detail of same in the stem, enlarged to show tumour tissue restricted to the procambial region and not extending into the cortex or pith. (Figs. 26 and 27 from sections communicated by E. J. Schwartz.)

Fig. 28. Section of dandelion leaf showing the large bundle on right infected with *Protomyces pachydermus* and that on left free. The fungus is confined to the phloem. (From section cut by W. R. I. Cook.)

PLATE XII.

Fig. 29. Tangential section of dandelion leaf infected with *Synchytrium taraxaci*, showing two large host cells, each surrounded by a capsule of small rectangular cells without intercellular spaces. In the angle of the vascular strand on the left is a surface view of an earlier stage of capsule formation around a host cell below the level of the section. At this depth most of the capsule comes from the mesophyll tissue. (From a section cut by W. R. I. Cook.)

Fig. 30. *Taphrina aurea* on poplar leaf. Early stage, showing elongation of a group of epidermal cells, one of which has divided.

Fig. 31. Normal fruit wall of passion fruit.

Fig. 32. Fruit wall of passion fruit affected by "woodiness," showing the increase of the normal layers of sclerotic cells and the transformation of the parenchyma below into stone cells.

Fig. 33. Later stage of "woodiness," showing the development of phellogen cutting off a sclerotic phelloderm below. (Figs. 31 to 33 from sections communicated by R. J. Noble.)

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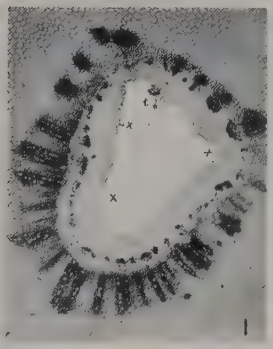
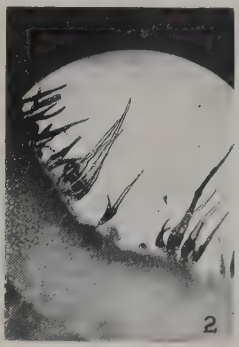
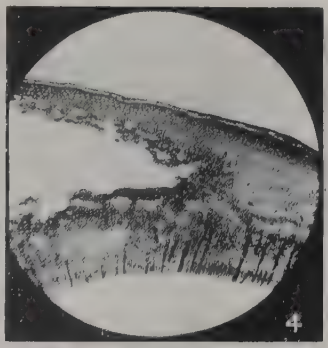
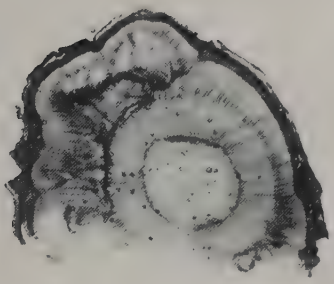
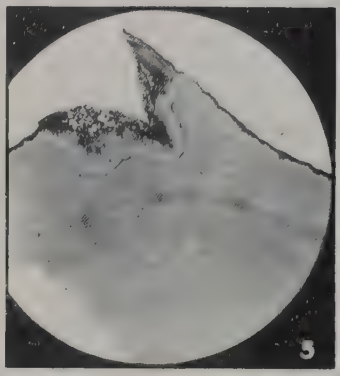
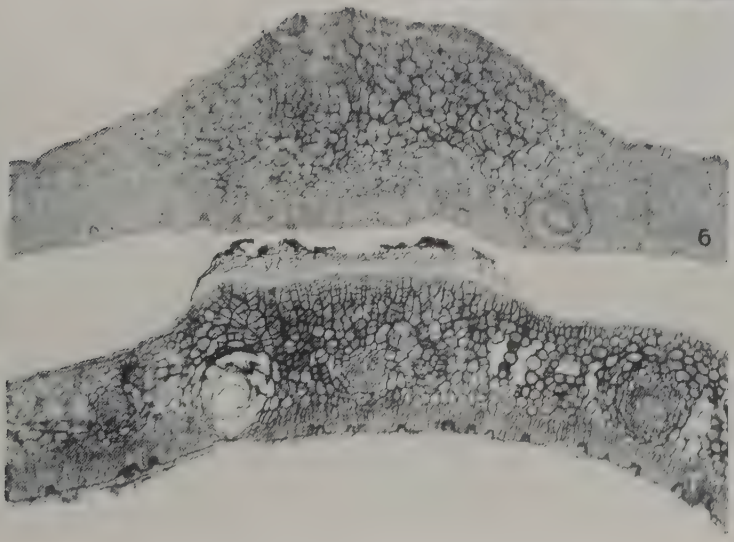




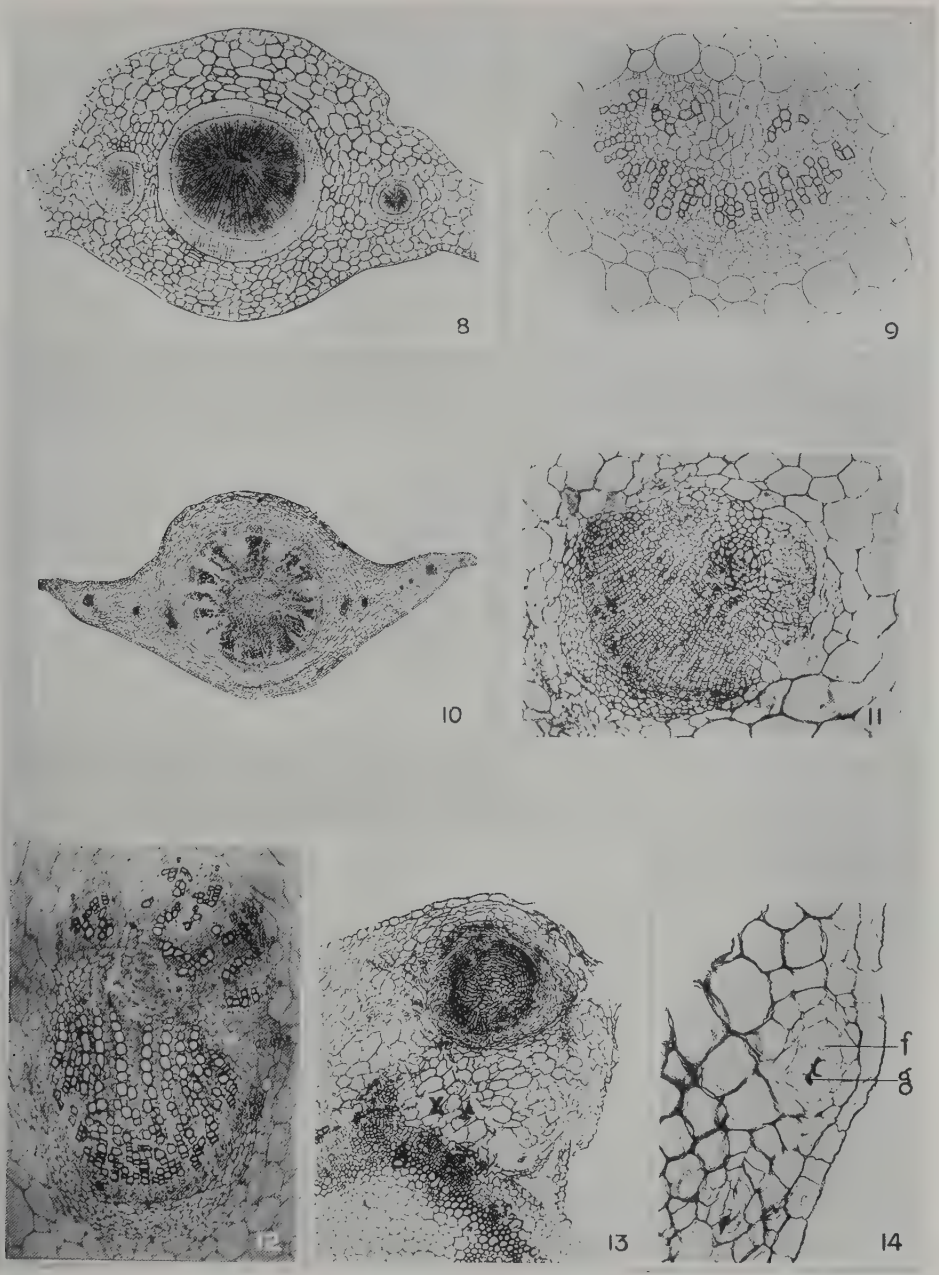








BUTLER.—Some Aspects of the Morbid Anatomy of Plants (pp. 175–212).



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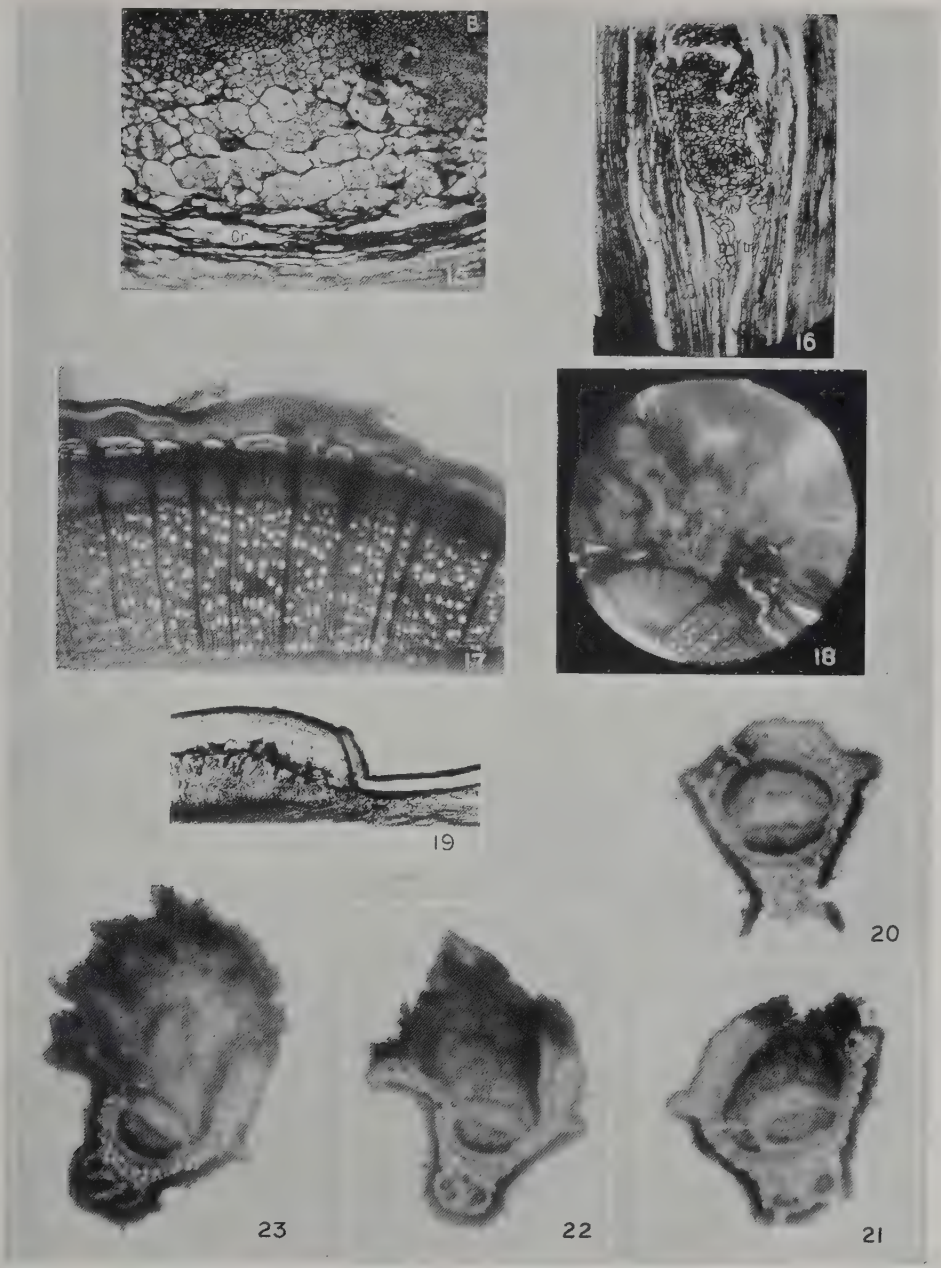

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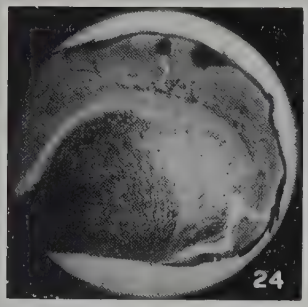

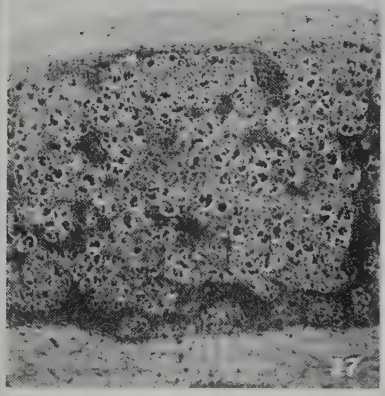

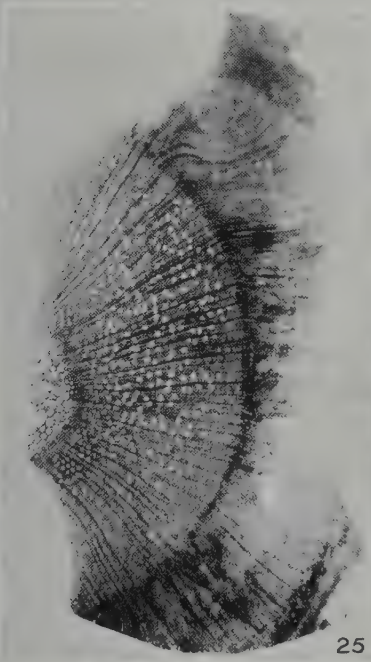

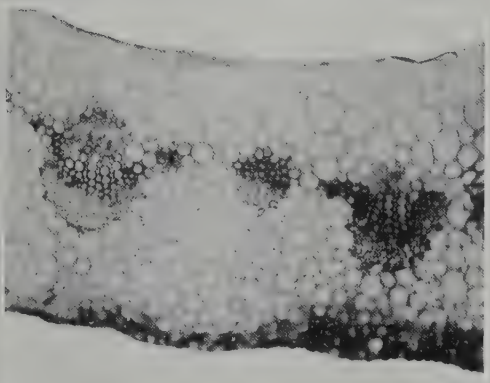

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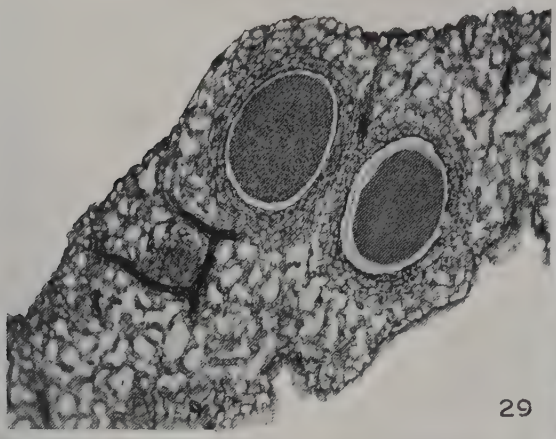

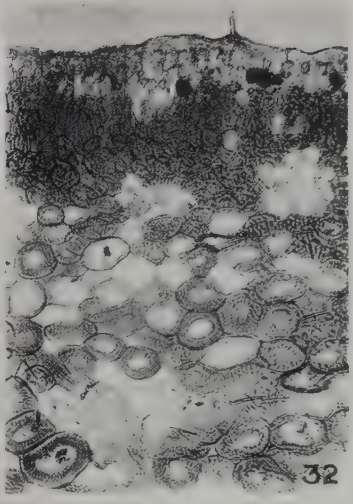

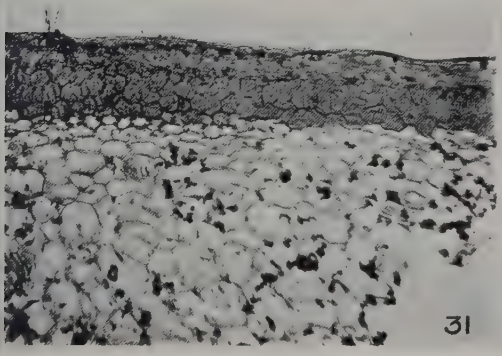

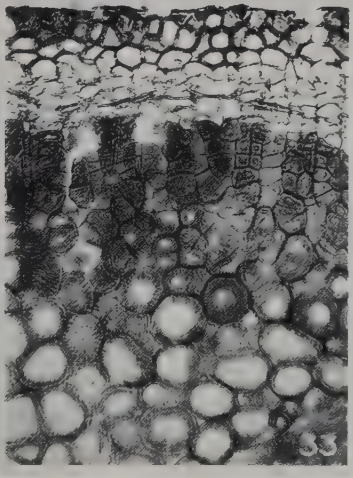

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Tropical Plant Diseases: Their Importance and Control*

By Dr. E. J. BUTLER, C.M.G., C.I.E., F.R.S.

IN the great tropical and subtropical plantation industries, such as tea, rubber, coffee, cocoa, citrus fruits, etc., large areas of permanent crops are cultivated on capitalistic lines with uniform and usually white control. On an estate of hundreds or even thousands of acres, often under highly intelligent supervision and where the produce may be worth a great deal of money, it is comparatively easy to get adopted improvements which are the results of scientific research, whether in the control of disease or in any other direction. It is quite a different matter, however, when one comes to deal with the crops grown by the indigenous people of the tropics for their own use. Their agricultural practices are rigidly traditional, their standard of intelligence may be low, money is scarce or even absent, and their crops are raised in small holdings, often subdivided to an almost incredible degree. I once had occasion to acquire 17 acres for expansion of an agricultural research station in India and found 30 families and 91 individual plots represented in this piece of arable land. In such conditions—and they are those under which a great part of the population of the world lives—"the cultivator's ways and the sheep's ways tend to be much the same", as an Indian proverb says, and however well the traditional agricultural methods are followed, the cultivator is apt to be helpless in an emergency as, for example, an outbreak of epidemic plant disease.

It is not surprising that the earlier plant pathologists who worked in the tropics, from the time when Marshall Ward went to Ceylon in 1880 to fight coffee leaf disease when it was already too late to save the industry from ruin, should have concerned themselves mainly with the diseases of the great export crops. A study of the reports of fifteen or twenty years ago will show that for practical purposes, India was the only tropical British possession in which it was the policy of the agricultural departments to devote much of their attention to the crops grown by the people of the country for their own use. During the past ten years, however, there has been a considerable improvement in this respect in the British Colonies, especially those of tropical Africa. Most of the colonies have one, and a few have two, plant pathologists attached to the agricultural departments and where as in several of them there are no plantation crops, the needs of the village cultivators are receiving attention. Even in some of the more advanced 'plantation' colonies such as Ceylon and Malaya, the large plantation industries have now organised their own research departments, leaving the Government departments of agriculture free to concentrate on the improvement of the local crops and methods of farming.

Such improvements are likely to increase the responsibilities of the plant pathologists. New and improved varieties of crop plants are liable to become attacked by diseases from which the old ones had become immune through age-long selection, and more intensive methods of farming often have a similar result. The great activity in crop improvement that has been characteristic of agricultural development in the United States and Canada since the beginning of the present century, has been accompanied by such an increased call on the services of plant pathologists that each of the staple crops has, not one but many, men engaged in the study of the cause and control of the diseases to which it is liable. As similar efforts are made to improve the staple food crops of the tropics similar needs will be felt. There are clear indications from the work on rice in Japan that even in a crop such as this, which in India is one of the healthiest of all the field crops, there is a number of diseases capable of becoming formidable obstacles to the introduction of improved varieties.

The two tropical cereals next in importance as food crops, sorghum and the bulrush millet, are much more subject to disease than rice, but very little work has been done on them in the tropics and even the full life-history of several of the common bulrush millet parasites is unknown. Still less is known of the diseases of the tropical pulses and other plants of economic importance, except those that are grown for export. Tropical plant pathology has not much to boast of in the study and control of village and field crop diseases; the number of man-years of work put into this branch of agricultural science is too small to have yielded much result and the difficulty of getting the native cultivator to change his ways, as well as his lack of means and general backwardness, has helped to induce a pessimistic outlook amongst those who are charged with the control of agricultural research and amongst the research workers themselves. Nevertheless, not only because there is great preventable loss of crops from disease in the tropics but also because a solid knowledge of the pathology of each crop plant is a necessary concomitant of all attempts at crop improvement, means must be found to surmount these difficulties. Little by little, openings for successful attack on them will appear, and however slow progress may be at first, the next fifty years are bound to see a great improvement in the crops and in the general agriculture of even the most primitive of the African Colonies. In this advance plant pathologists will have to bear an ever increasing share.

The work which has been done in India during

* From a semi-popular lecture delivered before Section K (Botany) of the British Association at York on Sept. 2.

the past twenty-five years or so illustrates some of the problems that the tropical pathologist has to face in dealing with village and field crops. When the Indian agricultural department was formed thirty years ago, extremely little was known of the diseases of tropical plants, though there were a few exceptions, such as sugar cane. The first work done in the mycological branch, therefore, was to make a survey of the diseases of the more important crops, and when many of these were found to be undescribed, a more intensive study of a few was undertaken and lasted a number of years. Two of them, one on palmyra and coconut palms and the other on rice, broke out in epidemic form and had to be dealt with on emergency lines, invoking the aid of the administrative services and leading, in the palm disease campaign, to legislation of the kind that is familiar in more advanced countries, where it becomes an actionable offence to own a diseased plant without reporting or dealing with it as prescribed. In the campaign against this disease, which was exterminating the palmyra in an area where this was the dominant tree and by far the most useful,* nearly a million diseased palms were cut out to save the rest. This and the subsequent legislative action and the discovery that many trees could be saved by removing the bud sheaths in the early stages of attack have been successful in preventing the spread of the disease and in keeping it within manageable proportions. The campaign cost the Government about £20,000; but the value of the palms cured by treatment was estimated at about £28,000 in 1921, and the number saved from infection must run into millions.

In other cases it soon became evident that the variety of the crop grown was exceptionally liable to disease either because of inherent susceptibility or because disease had been allowed to accumulate in the stock and was being transmitted when diseased material was used for planting each successive crop. In such cases the agriculturists of the department, each of whom had a district under his charge, became invaluable collaborators, both in observing the injury done to particular varieties and in introducing new or more healthy ones.

Then there were the cases where one had to make a direct attack on the parasite and try to kill it or to prevent its spores germinating by the use of fungicides. This at once brought one face to face with the economic limitations of village agriculture in countries like India, where the cultivator usually has little actual money at his disposal and can only borrow at exorbitant rates of interest.

Copper and sulphur have remained to the present day the chief weapons in the hands of the plant pathologists in their direct attack on fungal parasites. In India they have been used chiefly on the more valuable orchard and garden crops or as seed disinfectants on field crops.

* An ancient Sanskrit poem professes to enumerate eight hundred and one separate uses for the palmyra palm.

The relative infrequence of destructive epidemics of disease amongst indigenous crops in the tropics as compared with the great plantation crops is not due, I think, as is sometimes assumed, to the circumstance that the latter generally occupy larger continuous areas under the one crop, so that disease germs can multiply and infect more readily. In India there are large areas of village lands mainly under a single crop, as rice in 70 per cent of the cultivated land in parts of Bengal or cotton in 60 per cent of Khandesh. Freedom from disease in these cases is probably mainly due to disease resistance having become, through long experience, the determining factor in the selection of the varieties grown, the quality of which is liable to be a secondary consideration and is often decidedly low. In the plantation industries, on the other hand, quality which will enable the produce to compete in the markets of the world is so important that hardiness may be sacrificed. Examples of destructive epidemics in these crops are numerous and are not, as in the other category, usually due to newly introduced parasites. They are just as often due to an old parasite finding in a new variety a congenial host. They are sometimes also due to the considerable financial return leading to expansion of a plantation system into areas not naturally affording optimum conditions for the growth of the plant, so that its environment may become more favourable to the parasite than the host.

The recent wave of epidemic disease that has ravaged the cotton plantations of the Sudan may perhaps find a partial explanation in these considerations. When the great Gezira irrigation scheme, due in its inception to the genius of Garstin and Kitchener, was opened in 1925, it was already established that the highest quality Egyptian long-stapled cotton could be successfully grown in this previously arid waste. At the present time, approximately 200,000 acres of irrigated Sakel cotton is grown as the major crop in the gigantic plantation of 600,000 acres under uniform control and cropping, surely the largest arable farm in existence. During the first five years after the dam was built across the Blue Nile, cotton worth more than £12,000,000 was produced from the Gezira, and 150,000 people had settled where before there was only a scattered famine-stricken population.

The cotton disease known as blackarm occurs in all the cotton-growing parts of Africa, but it seldom causes much injury to the varieties grown by the people of the tropical regions, and the long-stapled kinds grown in Egypt escape damage apparently because the climate of Egypt does not suit the parasite. In the Egyptian varieties grown in the Sudan it became a formidable pest, being one of the main factors in reducing the yield per acre from 479 lb. in 1925–26 to 129 lb. in 1930–31. This represents a loss of more than 60,000,000 lb. of cotton on the area grown in the latter year, worth even at the low prices then prevailing, more than £800,000. As the disease is carried mainly

on the seed, an elaborate scheme of seed disinfection with the dust known as 'Abavit B' was carried through before the last season's crop was sown, the whole of the seed for this great area, representing more than 30,000 bags, being treated by specially devised seed dusters. Various other steps were also taken to combat the disease, and the yield, when harvest was completed last spring, was found to have risen again to a little more than 400 lb. per acre, or not far short of that of the earlier years.

In this case there is far more at stake than the saving of an industry, however important. The whole future of a province is in the balance. I cannot refrain from quoting a foreign observer who visited the Sudan two or three years ago. "The Sudan is the latest thing in European exploitation and it is the best." There has been "created a corps of agriculturists and entomologists to destroy the pests that attack the native crops; a group of veterinarians to look after and improve the native cattle; and a quite unrivalled body of biologists, bacteriologists, laboratory workers and doctors to fight native diseases. Trekking through the land are the government biologists and entomologists, experimenting, destroying pests, noting processes, giving advice. You rarely find them in the same place for two weeks running; these hardy scientists do even more work in the open than in their laboratories."

The West Indies present a very different picture from the Sudan. There, in some of the oldest of the British Colonies, generations of planters and settlers have been engaged in the tropical and subtropical cultivations of lands favoured by Nature to an unusual degree. Jamaica is the largest banana exporter of the British Colonies, having sent out 24,000,000 bunches in 1930, representing more than 50 per cent of the total value of her exports. In 1912 the first cases of the now notorious Panama disease of bananas in the island were examined by S. F. Ashby. Rigid quarantine measures were promptly introduced by the Director of Agriculture, Mr. H. H. Cousins, impressed by the ruin from this disease which had befallen the banana-growing enterprise in the Dutch Colony of Surinam between 1906 and 1910. As soon as a diseased plant was discovered, it and all the surrounding ones on an area of four chains had to be destroyed and the area fenced in. As a result, spread was slow, the number of cases annually not surpassing 1000 until 1921. Increase since has been at the rate of about 50 per cent a year, until by 1929, there were about 85,000 cases, involving nearly 140,000 plants. In the parish of Portland, the most diseased, some 9000 acres, or approximately one-tenth of the total estimated banana acreage of the island, have now been abandoned, for it has been found that commercial banana growing is impossible on land once infected, and the great United Fruit Company has already abandoned nearly 100,000 acres from this cause in Central America. The expenditure by the Government of some £60,000 in Portland no doubt prolonged the life of the plantations by ten years or more, but in the end has proved unavailing.

As there is no direct method of fighting this disease, which is due to a soil fungus, attempts to procure resistant varieties are being energetically carried out both in Jamaica and at the Imperial College of Tropical Agriculture in Trinidad. Varietal tests have shown that the Cavendish species of banana and some others are either highly resistant or totally immune. Botanists both from Kew and the West Indies have toured the world in search of varieties for trial and hybridisation and these are grown under quarantine and inspection at Kew before being sent on to Trinidad. Very large numbers of crosses have been made in Trinidad and Jamaica and some of the immune seedlings produced in the latter island have given bunches which were acceptable to the trade during the past year. The Trinidad seedling I.C.1 (Imperial College No. 1), a cross between the commercial Gros Michel and a wild species, has also shown remarkable immunity during six years tests, but the fruit still requires improvement.

It is estimated that within the next five to seven years, at the present rate of increase of the disease and the amount of suitable banana land left, the Jamaican export will begin to decline, and the decline is likely to be rapid unless a satisfactory resistant variety can replace the Gros Michel. The breeding work is difficult and slow. It is not easy for the stricken farmers to be patient. The whole population in the coastal parts of the parish of Portland has been brought up to the cultivation of the banana before anything else, and though an alternative crop of marketable value is desperately needed, it will take a long time to break down prejudices in a one-crop population. Many of the people have migrated, but many have remained to make a living as best they can. It is cold comfort to tell them, as there have not been wanting even scientific men to say, that these coastal lands, extraordinarily fertile though they be, are unsuited to the banana because the damp soil favours attack by Panama disease. The local Department of Agriculture has never taken that view but has striven hard to fight the disease, and the extra lease of life that it has given the industry, though insufficient in Portland, may yet save the banana cultivation of the island as a whole.

In the examples selected to illustrate the importance and control of the diseases of tropical plants, there is every gradation in severity from the sorghum smut which levies a moderate toll of about ten per cent of the crop on some millions of acres in India, to the Panama disease which completely exterminates the susceptible bananas and precludes replanting within any reasonable time. The success of the measures which have been adopted to control these diseases also shows every gradation from the complete control which is easy to obtain by disinfection against sorghum smut or by growing Cavendish bananas in Panama-diseased land, to mere alleviation which appears to be all one can hope for, but is yet sufficient,

against rubber mildew or the root diseases of limes. A consideration of these measures—ruthless eradication, the complete replacement of susceptible varieties, hybridisation or selection to obtain resistant plants, budding or grafting on resistant stocks, modifications in agricultural practices like stubble burning or pre-watering, and, finally, the direct attack on the parasite by steeping, spraying or dusting—indicates how varied is the task of the plant pathologist and how wide must be the foundations of his knowledge if he is to perform it successfully. The old conception of the mycologist, student of the fungi that cause disease, as adequately equipped to fulfil the duties of a plant pathologist, dies hard but it is dying. Like bacteriology in the realm of medicine, pure mycology is a necessary foundation and the mycologist a necessary collaborator, but he is not equipped either as a general practitioner or a specialist in particular diseases.

The practical man is often slow to admit that a destructive disease in a plant is due to agencies outside his control. Confronted with such he is inclined to seek for explanations other than the true one. He looks first for some disorder brought about by cultivation or inbreeding, or meteorological phenomena. Or he thinks that the soil is unsuitable or has become exhausted, or that the plant, if an exotic, has failed to become acclimatised. It is often not until all these have been tested and found wanting, that the true cause is fully realised. Experience has shown that it is unfortunately rare to find the explanation of serious disease in these directions and the dominating factor is usually the presence of a parasite, however much its activities may be favoured by secondary causes.

Failure to recognise the very varied weapons used by modern plant pathology and undue weight given to the secondary factors in the causation of disease have led, no doubt, to the suggestion which I have heard that the 'mycologist', as he is still called in most of the British Colonies, may be in danger of losing his position as one of the most essential members of any team of tropical research agriculturists. In actual fact there can be no question that, looked on as a member of a team and relying on the collaboration that must exist between him and the plant breeders and agriculturists, the plant pathologist is more needed now than ever. Improvement in the crops of the people and the quest of quality or the satisfaction of market fancies in the plantation crops can be relied on to be followed by increase in disease. Unless plant pathologists well versed in the pathology of the crop concerned are available—and they cannot be produced at a moment's notice—the examples I have given will be paralleled in every Colony in the British Empire.

The Cambridge Philosophical Society

THE Cambridge Philosophical Society was incorporated in 1832, by Royal Charter, through the grace and favour of William IV, and the centenary of that event has been celebrated recently. (See NATURE of November 19, p. 769.)

The history of this scientific organisation, which has maintained a high standard in past years, and remains unfailingly hopeful of the future, should be of interest, not only to historians of science, but also to laymen. The Cambridge Philosophical Society is of older origin than its royal grant by some twelve or thirteen years. In October 1819, as the outcome of conversations amongst a few interested persons, a notice was issued, signed by thirty-three members of colleges, inviting assistance in promoting a society "as a point of concourse for scientific communications". Success having followed this venture, the designated name was approved, rules were made, and a council elected for the year ensuing. The Chancellor of the University, H.R.H. the Duke of Gloucester, accepted the office of patron; that of president being assigned to the Rev. William Farish, Jacksonian professor of natural and experimental philosophy. In all the foregoing movements, Prof. A. Sedgwick, J. S. Henslow (afterwards Prof.), and Dr. E. D. Clarke (professor of mineralogy, 1808–22), were prominent. At an inaugural gathering, the last-named gave an address on the new project (separately distributed*). Reference was broadly made to the philosophical contributions of members of the University as being hitherto "frittered and squandered away" in detached and distant parts, almost to be without existence.

The first half of the nineteenth century witnessed the establishment of many scientific societies. The apprehensions of Sir Joseph Banks for the welfare, even the continuity of the parent tree, the Royal Society, were seen to have been needless, and indeed, at his death, in 1820 (whilst in office), however chequered in progress certain of the newcomers were, the writing was already on the wall in regard to alarmist notions. Among the new bodies were: the Horticultural (1804), Geological, London (1807), Geological, Penzance (1814), Institution of Civil Engineers (1818), Astronomical (1820), Zoological (1826), Geographical (1830), Entomological (1833), Chemical (1841). Cognate organisations were formed at Manchester, Glasgow, Hull, and other centres. Neither in the perplexing era of the seventeenth century, nor in the industrial nineteenth, were scientific societies born secure in the knowledge of powerful support. In all the instances above, initiation was *ex collegium*. In the case of the Cambridge Society, there certainly was an ample measure of University support and

*The Patent Office Library, London, possesses a copy (bound) on the title page of which is the pencilled signature, "J. Henslow, 1821," and there are textual annotations in the same hand.

Les fausses analogies entre le « Crown-Gall » des Plantes et les tumeurs malignes des Animaux

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L'analogie qu'on a cherché à établir entre le *crown-gall* des plantes, dû au *Bacterium tumefaciens*, et les tumeurs malignes des animaux repose en partie au moins sur les considérations suivantes :

1° *L'existence de métastases dans le crown-gall.* — Lorsqu'on inocule le *B. tumefaciens* dans les tissus jeunes des tiges des plantes, des galles secondaires apparaissent parfois à une certaine distance du point d'inoculation. E. F. Smith avança que ces galles résultaient d'une intrusion des cellules de la tumeur dans l'axe de la tige, qui de là gagnaient les pétioles. Ces faisceaux si caractéristiques de certaines formes de *crown-gall* étaient formés d'un cœur de cellules dérivant directement de celles développées au point d'inoculation et présentant les caractères morphologiques héréditaires du tissu constitutif de la galle primaire. Ainsi les galles secondaires ne seraient que des intrusions de la galle primitive.

Cette interprétation s'appuyait sur la similitude d'arrangement des faisceaux des tumeurs et des tiges, même quand la traînée a gagné le pétioe·

Mais Winkler a montré depuis longtemps que, si l'on enracine un pétiole, les faisceaux vasculaires y prennent la disposition caractéristique de celle de tiges, et il est facile de voir sur les photomicrographies de Smith qu'il en est de même dans les faisceaux du *crown-gall* qui se développent *in situ* et ne résultent en aucune manière d'une intrusion partie d'une tige.

De même Robinson et Wlakden en Angleterre, Riker aux États-Unis ont montré, chacun de son côté, en 1923, que ce qu'on appelait les métastases du *crown-gall* résultait de deux processus très simples et bien différents de ceux qu'on observe dans le cancer.

Le premier était l'élongation de la partie où a été faite l'inoculation dans les plantes à croissance rapide, l'inoculum montant avec les tissus ascension accompagnée de formation de galles secondaires à une distance considérable au-dessus du point d'infection. Le second de ces processus était le passage des bactéries le long des cavités intercellulaires longitudinales ; les galles secondaires résultaient donc de ce que l'agent pathogène passait par ces lacunes, déterminant la formation d'une nouvelle tumeur aux pointes de multiplication active. Pas plus dans un cas

que dans l'autre il n'y a intrusion du tissu de *crown-gall* dans les parties saines adjacentes. Smith lui-même, dans ses communications ultérieures, était bien près d'en convenir. Les cellules du *crown-gall* elles-mêmes, comme celles de toutes les autres galles végétales bien étudiées, se forment par division des cellules de la plante hospitalière, la galle résultant de l'accumulation des produits des cellules normales ainsi développées. Dans le *crown-gall*, une seule cellule peut, en se divisant, produire jusqu'à cent cellules, tandis que, dans certaines entomocécidies développées sur les tiges, la prolifération peut atteindre quarante cellules ou plus.

2° *Ce qu'on a appelé les embryomates du crown-gall.* — L'assimilation nd *crown-gall* aux cancers des animaux a paru justifiée par le développement à la surface de la galle de feuilles ou de pousses feuillées, en des points où normalement aucune formation similaire n'intervient. Ces formations ont été rapprochées par Erwin Smith des embryomates. Mais ces productions peuvent être consécutives à un traumatisme ou se produire à la suite de greffes hétérogènes entre espèces éloignées, par exemple dans les Solanacées. Elles correspondent au cal des boutures et se développent surtout quand la surface de la section en voie de cicatrisation reçoit une nourriture excessive.

En résumé, l'étude de l'anatomie pathologique du *crown-gall* ne justifie aucunement l'assimilation qu'on a voulu établir avec le cancer des animaux. La galle résulte toujours de l'entassement de couches cellulaires formées par divisions successives, comme dans les autres galles végétales (myciocécidies ou entomocécidies).

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COTTON DISEASES

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Cotton resembles wheat in the difficulty it presents of assessing the losses in yield caused by cryptogamic and virus diseases, but while methods have been devised for determining the damage done by most of the major wheat diseases, nothing of the kind is available, so far as the writer knows, for cotton. A few of the diseases mentioned below, Texas root rot and some types of *Fusarium* wilt, for instance, present no great difficulty, for they kill groups of plants and leave bare patches of greater or lesser extent in the field. When *Fusarium* wilt is sporadic, however, so that only single plants wilt in a scattered fashion throughout the field, the amount of loss of crop depends on the age at which the plants die and the habit of the variety grown. In this type of wilt in parts of Khandesh in India, the writer concluded that there was little loss when the plants wilted before they were half grown unless the incidence of the disease exceeded about 10 per cent. of the stand. Khandesh cotton branches freely, and the gaps left by the death of young plants were largely filled up by the surrounding ones. Had a cotton of the Sakel habit been grown, this would not have occurred, as this type does not form sufficiently extensive laterals to fill up bare spaces. In the neighbouring area of the Berars, *jari* cotton is killed by wilt in large patches which are left quite bare, and the loss may be heavy and is easily estimated. With other diseases it is often exceedingly difficult to assess the damage, either because other causes may be concerned in addition to the disease (climate or soil factors, for instance), and the extent to which they are responsible for increasing the loss of crop may not be calculable, or because the disease does not kill but merely weakens, and a method of determining the effect of this on the total crop has not yet been evolved. Examples of the former are found in blackarm and of the latter in most of the leaf diseases of the crop.

It will be understood, therefore, that the estimates that have been framed of losses from disease in the cotton crop allow of a considerable margin of error. The most elaborate are those of the United States Department of Agriculture as given in Supplement 83 of the *Plant*

Disease Reporter, 1932. They cover the three years 1928, 1929, and 1930, but are incomplete, for several States in the cotton belt furnished no returns. For those that did report, comprising about three-quarters of the cotton belt, the total loss from diseases (other than injuries caused by insects, worms, etc.) is given as about 16 per cent. of the crop. This represented an average loss of over 1,600,000 bales of cotton annually from three-quarters of the cotton belt during the three seasons in question, or assuming that the non-reporting States had equal losses, disease reduced the United States cotton crop annually during these years by well over 2,000,000 bales. Individual State estimates may indicate losses even greater than this, Florida estimating in 1930 that the average annual loss was somewhere between 15 and 25 per cent., Texas counting its loss from a single disease, the root rot, at 10 to 15 per cent., and Arizona calculating that the same disease costs the farmers in the State (not a large cotton grower) up to $5,000,000 in some years.

In the Russian province of Azerbaijan, bordering the Caspian Sea, Jaczewski states that in 1927 wilt reduced the yield by 9·1 per cent. In Russian Turkestan, Zaprometoff in 1929 estimated that some 150,000 acres of cotton are infected by wilt which reduces the crop by 27 per cent. Fahmy made some estimates of the prevalence of the same disease in some villages in Lower Egypt in 1924, and cites a case, not, he says, an uncommon one, where 25 per cent. of the Sakel plants on an area of 644 feddans (out of 696 feddans of Sakel grown by the village) had wilted, estimating the loss of crop at about one-sixth. In 1927 he reported that wilt had become so severe in some of the best cotton lands of the Delta that many cultivators had abandoned the susceptible Sakel for hardier (wilt-resistant) short staple varieties. In 1930 he estimated that the loss due to wilt in the Sakel crop of Lower Egypt varied between 5 and 15 per cent. annually.

The third in importance, after root rot and wilt, of the major diseases of cotton is probably the bacterial disease due to *Bacterium malvacearum*, which causes blackarm, angular leaf spot, and bacterial boll rot. In 1927 it was estimated to have caused a reduction in yield in the United States of approximately 500,000 bales, and in the West Indies it is said to destroy from 2 to 20 per cent. of the crop. In the Sudan the losses in certain years in the Sakel crop have been undoubtedly higher than those recorded elsewhere, but it is probable that few would be prepared to attempt an accurate estimate of them.

Anthracnose and sore-shin are two diseases that must be included as major affections, for in certain places and under certain conditions

they can cause great injury to cotton. Anthracnose was stated in 1930 to cause an annual loss in the southern States of about 150,000 bales on an average, but to vary greatly from year to year. Losses of 90 per cent. of the bolls from this cause have been recorded from some of the Florida cotton fields, and in 1923 it caused an estimated loss of 36,000 bales in North Carolina. Sore-shin is well known as the cause of much trouble in Egypt when sowing is done under certain weather conditions, and is stated to be the most common cause of poor cotton stands in Florida. Severely affected fields are usually resown, and hence it is impossible to assign figures to the losses caused by this disease, but it is always a serious problem in places where the cotton is liable to be sown in cold soils. In warm soil conditions it is incapable of causing injury.

Another disease of considerable importance, but for which it is difficult to give figures, is the internal boll disease, due to several allied yeast-like fungi that are conveyed into the bolls by cotton stainers (*Dysdercus*) and the green bug (*Nezara viridula*). Sea Island cotton in St. Vincent and Montserrat frequently lost most of the late bolls from this cause before control measures were taken in hand. In Wagale cotton an incidence of 7·5 to 17 per cent. has been reported from Burma and one of 24 per cent. in Cambodia cotton.

A disease, the full potentialities of which are not yet known, but which may come to be considered one of the major diseases to which the cotton plant is liable, is the virus disease known as leaf curl, which has hitherto been found only in Nigeria and the Sudan. Its restricted incidence, however, keeps it from being ranked as a major cotton disease at the present time, and there are no reliable estimates of losses from it known to the writer.

Minor diseases of the leaves and bolls are numerous, and almost any of them can at times and under certain conditions cause a good deal of injury. Most of these, though in the aggregate they must cause an appreciable loss of cotton, are seldom considered of sufficient importance to justify attempts to estimate the injury they do.

Of the major diseases mentioned above, the Texas root rot is the most remarkable in many ways. It is confined to the south-western part of the cotton belt and is not known anywhere else except for reports from Russian Central Asia. It is caused by a soil fungus, *Phymatotrichum omnivorum*, a natural denizen of the virgin soils of this region and one which is capable of attacking the roots of a very large number of plants, from lucerne to fruit trees and wild bushes: about 550 of these host plants have been recorded. The total annual

money loss caused by this fungus on all crops in Texas was put at the almost fabulous figure of $100,000,000 in 1931, and at the Fourth Annual Conference, held in that year, of the workers engaged on this disease jointly by the Federal and Texas authorities, eighteen of the members present were giving their whole time to its study and another sixteen were part-time workers on it.

The fungus spreads like a fairy ring through the soil, killing the plants at the margin of spread each season, but tending to die out in the centre of the circle. On the surface of the soil or the side walls of trenches, etc., it forms spore-bearing mats, but most of its life is passed underground, and it is well equipped by the possession of resting sterile stages (sclerotia) and mycelial strands to dispense with the ordinary means of spread by air-borne spores. Indeed, inoculations with the spores have not been successful and they are not known to play any part in disseminating the disease. It is most prevalent on alkaline soils and has not been found on those of a definitely acid reaction, being rather in contrast to wilt in this behaviour. It passes the winter on old cotton roots or on various wild plants and weeds, a wild convolvulus, *Ipomœa trichocarpa*, being one of the worst offenders in this respect.

Rotation, with such an omnivorous feeder, is not much use unless bare fallows can be maintained or non-susceptible crops (*e.g.*, cereals) be grown and kept clean weeded for several years. Trenches and other barriers to check the steady advance through the soil are effective if made to a depth of two to four feet, but are naturally expensive. It has been found that sorghum acts as a barrier to spread, and belts of four to twelve rows of grain sorghum kept free from weeds have proved valuable in saving uninfected fields from spread underground from adjacent infected ones. Finally, attempts have been made to find strains of cotton resistant to root rot so that they might be used for breeding or selection of suitable resistant stocks, but so far without much success. Altogether the root rot is a most formidable problem.

Cotton wilt is one of a large group of plant diseases characterized by invasion of the water-carrying vessels of the root and stem by a parasitic fungus. Until recently it was believed that only a single species of parasite was concerned, *Fusarium vasinfectum*, but it is now known that several other species of *Fusarium* can attack cotton roots in Egypt and the Caucasus, and that a very similar disease is caused by quite another kind of fungus, *Verticillium albo-atrum*, in the United States, South Africa, Central Asia, and Bulgaria. All these fungi produce similar symptoms, and the term "wilt" may apply to all. They are soil organisms and extremely persistent, so

that it is difficult to exterminate them once the soil becomes infected.

Penetration into the plant occurs through the fine roots, but once the fungus reaches the water-conveying vessels it spreads along these and may extend up the stem to a considerable height. The wilting is due partly to the blocking of the vessels and partly to a toxic action of excretions from the fungus. The leaves turn mottled or yellow, droop, and die, and the plant dries up. On the stems, especially towards the base, the fungus comes to the surface as the plant dies and produces pinkish masses of spores which disseminate the disease. Spread also occurs through the soil so that spreading patches, in which at times all the plants are killed, result.

Control is by prolonged rotation and the growing of resistant varieties. Fortunately a number of these latter are known, and wilt-resisters have now been developed that are suitable for most conditions. These are in wide use for wilt-infested lands in the United States, and both in Egypt and in India several resistant types have been selected and are now being pushed. As is usually the case, it has been found that each country must produce its own wilt-resisters. Fortunately, true immunes appear to occur in several of the main cotton types—*e.g.*, the Ashmounis in Egypt, Buri (*Gossypium hirsutum* var.) in India, and the Bokhara var. of *G. herbaceum* in Russia (against the wilt prevalent in Azerbaijan due to *F. caucasicum*). True immunity does not seem to have been encountered in American Upland cottons in the United States, but a number of varieties, such as Dixie and its derivatives, Miller, Willis, Cleveland 54, etc., are commercially resistant or tolerant and give good crops in wilt-infested land.

There is no need to refer other than very briefly to the bacterial disease, in view of the series of papers by Massey, Hewison, and others in this Review on the outbreaks of blackarm in the Sudan during recent years. The organism occurs in most cotton-growing countries, but is only severe in the Sudan, some parts of the United States, the West Indies, Nigeria, Guatemala, South-Eastern Russia, and Korea. The long staple Egyptian and Sea Island varieties are the most susceptible to its attacks, and this is one of the principal reasons why Egyptian cotton has not been profitably grown in the south-eastern parts of the United States. Nevertheless, cotton in Egypt itself does not suffer appreciably from *B. malvacearum*, though the organism is common there and the same cotton, Sakel, as in the Sudan, is widely cultivated. This fact shows its close dependence on its environment as a source of harm. High temperature and high humidity have been

proved by work both in the Sudan and at Rothamsted to be most suitable for successful infection, but exceptions have been reported, such as a dry weather outbreak of blackarm and boll rot, with little or no leaf spotting, in Barbados in January, 1918.

Seed treatment is the most effective direct method of control, as the organism is freely carried over from one crop to the next on the seed. Soil infection is considered in the United States to be infrequent because the organism does not remain long alive in the soil, but the experience in the Sudan suggests that infection frequently comes from the soil (dust, etc.) from the previous year's fields, so that adequate crop rotation and distance from the fields that bore the previous crop may be important. Resistance is known to occur in certain strains, all the pedigree strains of Sea Island now grown in St. Vincent showing considerable resistance to the disease.

Anthracnose, caused by the conidial (*Colletotrichum gossypii*) stage of *Glomerella gossypii*, used to be considered the most serious cotton disease in the United States before root rot and wilt became prominent. The fungus does little or no harm to the growing plant though it sometimes causes damping off of the seedlings, but it attacks the bolls through the flowers, or more usually on the side walls, causing sunken spots that often penetrate to the lint and seeds and that become covered with pinkish spore masses. The effect is to dry up or distort the bolls and ruin the lint, sometimes only in one or two of the locks. The organism is very widely distributed in the New World; India and the Sudan appear to be mostly too dry for it, but it occurs in Burma and is prevalent throughout most of West and West-Central Africa. Anthracnose frequently follows bacterial boll rot and may obscure the latter.

As the disease is transmitted on the seed, the use of clean seed is usually sufficient for control. It has been found not to survive on the seed for much more than a year, so that two-year-old seed is usually quite safe. Machines for the hot-air treatment of cotton seed against this disease have been devised in America, but with reasonable rotation and two-year-old seed their use is not necessary.

Sore-shin due to *Rhizoctonia* (*Corticium*) *solani* occurs everywhere, this fungus being a regular inhabitant of most soils in all countries. In classical studies carried out in Egypt many years ago, Balls showed that it can only cause injury to the young cotton when sown in cool soils and kept cool during the very young seedling stage. In this its behaviour is the reverse of blackarm, and the relative prevalence of the two diseases in Egypt (sore-shin common, blackarm rare) and the Sudan (the reverse) reflects this difference in their temperature

preferences. Early sowing to avoid boll weevil injury in the States has been considerably hampered by increased tendency to sore-shin. Seed treatment with dusts such as Abavit and various other mercury-containing fungicides has reduced damping-off due to this and other fungi, but the best method where possible is the avoidance of sowing in cold, damp soils.

Besides the killing of seedlings due to this fungus various other types of damping-off occur in the seedling crop. A frequent form in India (known also in Uganda, Nigeria, Trinidad, and Egypt) is due to *Macrophomina phaseoli*, another common soil fungus, but one apparently confined to warm countries. Its attacks cause the seedlings to dry up while still erect, and are more sporadic than those of the true sore-shin fungus, though a mortality of 30 per cent. has been seen at times in the Central Provinces of India; no way of avoiding them is known. *Gibberella moniliformis* causes a root rot of seedlings in the southern States (especially Georgia) and in Turkey, and has also been found in Tanganyika, Uganda, Queensland and elsewhere injuring the bolls, usually such as have been punctured by insects. Species of *Phytophthora* and *Pythium* have also been reported to cause damping-off in India, and *Sclerotium rolfsii* in the West Indies, India, Ceylon, Porto Rico, and Texas, the latter being particularly troublesome in wet weather on Sea Island cotton in St. Vincent.

The internal boll disease is one of the most remarkable of fungal diseases, being the first that was studied of a widespread type of fruit diseases due to a curious group of fungi that are introduced into the interior of the fruit by sucking insects. They have been given the special name of Stigmatomycoses. The stigmatomycosis of cotton is due to one or other of five of these fungi, *Nematospora gossypii, N. coryli, N. nagpuri, Spermophthora gossypii*, and *Eremothecium cymbalariæ*, of which the first two are the most widely distributed, being found in the West Indies, South Africa, most of tropical Africa (the Gezira tract in the Sudan being an exception), Russian Central Asia, and Burma. *N. nagpuri* has so far only been found in India, and *S. gossypii* and *E. cymbalariæ* in the West Indies. As, however, their presence is often masked by secondary followers, their distribution is probably much wider than reported hitherto. *N. coryli*, for instance, has recently (1928) been found on cotton in California, and is quite likely to occur on this host also in the eastern States.

All these fungi have minute needle-shaped spores with, in addition, round or oval yeast-like cells. These spores are introduced into the interior of the bolls in the sucking apparatus of the *Dysdercus* or

Nezara, and no sign of their presence is visible until the boll opens, when the lint is found stained and rotted and the seed often shrivelled. The fungi are active fermenters and appear to be able to penetrate through young cell walls and unthickened lint fibres. In the seed, penetration into the embryo has been observed, but the exact relation of the fungi to the host cells appears to be still obscure. It is believed that the insects pick up the fungus from other host plants in the neighbourhood of the cotton fields, and in South Africa the tree *Sterculia platanifolia* was implicated in a subsequent outbreak on the cotton fields. In Uganda, where this is regarded as the most serious cotton disease, it has been shown that the fungi cannot be transmitted by young *Dysdercus* nymphs, nor grow through the boll wall even if wounded. In the West Indies and Nigeria they are held to be responsible for considerable boll shedding, and in East and South Africa and Burma this must rank as a major disease of the crop.

Excellent results were obtained in St. Vincent by the eradication of the alternate food plants of *Dysdercus* that are utilized by the insect early in the season. This so delays the appearance of the stainers that a clean crop may be harvested. It is probable that this is the most hopeful line of attack on a disease that is exceptionally difficult to control.

Leaf curl was first seen in Nigeria but did not attract much notice until its appearance in epidemic form in the Sudan in the last few years. Space does not permit of an account of this disease, which has been fully described in several recent publications. It differs from all the others mentioned in being due to an invisible agent or virus, but resembles the internal boll disease in being insect-transmitted, the vector being the whitefly, *Bemisia gossypiperda*. Control will ultimately come from the growing of resistant varieties, several of which have already been found amongst the long and short staple cottons available locally, but until one suited to the special conditions of the Gezira is developed, every effort is being made to prevent the disease from being carried over the period between successive crops by eradicating self-sown and ratoon cotton after harvesting.

Some of the minor diseases of cotton can cause considerable injury in certain conditions. There is, for instance, a soft rot of the bolls in St. Vincent, Montserrat, and Porto Rico due to *Phytophthora palmivora* and *P. parasitica*, which destroyed over 60 per cent. of the bolls in July plantings in St. Vincent in 1927, but which can be largely avoided by late planting (after September 1). The boll disease due to *Diplodia gossypina* is said to render 50 per cent. of the bolls worthless in many fields in Florida and to have cost the State as a whole

about 20 per cent. of the crop in the wet season of 1928. Evidence has been brought forward in the United States to show that this fungus is an imperfect stage of *Physalospora rhodina*, and, if so, it belongs to one of the most widely distributed tropical weakly parasitic fungi. But like various other boll rots attributed to common moulds such as *Rhizopus nigricans* and other species of this genus, *Aspergillus niger*, *Alternaria*, *Fusarium*, *Cephalosporium*, *Epicoccum purpurascens*, and *Nigrospora oryzæ*, these are so likely to follow and obscure more primary boll-rotting organisms that much caution is required in considering them as the real cause of injury. This does not apply, however, to two species of *Alternaria*, *A. macrospora* and *A. gossypina*, which cause leaf spots and boll rots in Nigeria, Southern Rhodesia, Uganda, Tanganyika, and Trinidad. This disease can cause heavy defoliation and boll shedding in wet weather in Nigeria, and the fungi have been proved experimentally to be true parasites of cotton.

Of the large number of minor leaf parasites there are a few that seem to have dangerous possibilities, such as the mildew *Erysiphe malachræ* common in the West Indies and Peru, the rust *Cerotelium desmium* found almost throughout the cotton-growing countries, the leaf spots due to *Ascochyta gossypii* and *Mycosphærella gossypina*, and the areolate mildew *M. areolæ*. All these true parasites occur chiefly on mature leaves or too late in the season to do much harm, but serious injury has been recorded somewhere from all of them, and any may one day become important. Phytopathological history is full of records of parasites long regarded as harmless but that have proved to be the limiting factor in the introduction of some new improved variety or in the extension of the crop into some new region.

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NOTE

ON THE INCIDENCE OF CACAO DISEASES IN THE BRITISH COLONIAL EMPIRE AND THE STEPS BEING TAKEN TO INVESTIGATE AND CONTROL THEM.

I. — *Canker and black pod caused by Phytophthora palmivora.*

This, which is probably the most serious cacao disease throughout the world, occurs in all cacao-growing regions of the British Empire and causes heavy losses in the West Indies, West Africa, Ceylon, and elsewhere. It was first investigated in Ceylon and its cause was discovered in Trinidad. Research work on the conditions influencing its attacks is being carried out on an extensive scale in the Gold Coast, where the Mycologist of the Department of Agriculture, Mr. H. A. Dade, is devoting almost his whole time to the study of cacao diseases and has published a series of papers on them in the publications of the local Department of Agriculture. This and other cacao diseases are also discussed by R. H. Bunting, formerly Mycologist of the Gold Coast Department of Agriculture, in Proc. Ist W. African Agric. Conf., pp. 86-97, 1927 and Gold Coast Dept. of Agric. Bull. 11, 1928 (the latter illustrated by coloured plates). The control of this disease by spraying with Bordeaux mixture was shown to be practicable many years ago in Trinidad, but spraying is not now known to be carried out anywhere and Dade is advocating early removal of affected pods and general sanitary measures (Gold Coast Dept. of Agric. Bull. 23, pp. 109-128, 1931). The later estimates that at least 20,000 tons of cocoa, worth about £ 200,000, are lost annually in the Gold Coast by pod diseases, of which this is the chief.

II. — *Witch broom caused by Marasmius perniciosus.*

At present confined to South America and Trinidad, this disease has been mainly responsible for the decay of cacao growing in Dutch and British Guiana and is threatening that in Trinidad, where it is under investigation by the Mycologist of the Department of Agriculture, Mr. F. Stell, and by the staff of the Imperial College of Tropical Agriculture. The progress of these investigations is recorded in the recent annual reports of the Department and in the journal, «Tropical Agriculture,» issued by the College. Mr. Stell has visited Ecuador (where the disease is destructive) with a view to obtaining possible resistant varieties, and the Colonial Advisory Council for Agriculture and Animal Health has recently recommended the despatch of a botanist for the same purpose to Brazil and other areas where resistant varieties may

NOTE

RELATIVE AUX MALADIES DU CACAO DANS L'EMPIRE COLONIAL BRITANNIQUE ET AUX MESURES PRISES EN VUE DE LES ÉTUDIER ET DE LES COMBATTRE.

I. — *Chancre noir des cabosses provoqué par le Phytophthora palmivora.*

Cette maladie, qui est probablement la maladie la plus sérieuse du cacao dans le monde entier, apparaît dans toutes les régions de l'Empire britannique qui s'adonnent à la culture du cacao et provoque de lourdes pertes aux Indes occidentales, en Afrique occidentale, à Ceylan et ailleurs. Elle fut d'abord étudiée à Ceylan et la cause en a été découverte à La Trinité. Les recherches relatives aux conditions qui influencent les attaques de la maladie sont poursuivies sur une grande échelle à la Côte de l'Or, où le mycologiste du Département de l'Agriculture, Mr. H. A. Dade, consacre presque tout son temps à l'étude des maladies du cacao et a fait paraître une série d'articles à ce sujet dans les publications du Département local de l'Agriculture. Cette maladie du cacao, ainsi que quelques autres, font également l'objet d'études publiées par R. H. Bunting, ancien mycologiste du Département de l'Agriculture de la Côte de l'Or, dans le compte-rendu de la première conférence agricole de l'Afrique occidentale (pages 86-97, 1927), ainsi que dans le bulletin nº 11 de 1928 du Département de l'Agriculture de la Côte de l'Or (ce dernier article contient des planches en couleurs). Il y a quelques années, cette maladie était combattue efficacement à La Trinité au moyen de la bouillie bordelaise, mais actuellement, ce procédé n'est plus employé nulle part et Dade conseille d'enlever à temps les cabosses malades et de recourir à des mesures sanitaires générales (bulletin nº 23 de 1931 du Département de l'Agriculture de la Côte de l'Or, pages 109-128). Ce dernier estime que les maladies des cabosses, dont celle examinée ici est la principale, occasionnent à la Côte de l'Or une perte annuelle d'au moins 20,000 tonnes de cacao, représentant une valeur d'environ 200,000 livres sterling.

II. — « *Balai de sorcière* » *provoqué par le Marasmius perniciosus.*

Actuellement confinée dans l'Amérique du Sud et à La Trinité, cette maladie est la cause principale du déclin de la culture du cacao dans la Guyane hollandaise et anglaise et elle menace celle ds La Trinité, où elle fait l'objet d'études entreprisee par Mr. F. Stell, mycologiste du Département de l'Agriculture, ainsi que par des agents du Collège impérial d'agriculture tropicale. Les progrès réalisés par ces études sont consignés dans les rapports annuels récents du dit Département, ainsi que dans la revue « Tropical Agriculture », éditée par le susdit collège. Mr. Stell a visité l'Equateur (où la maladie est destructive) en vue d'obtenir éventuellement des variétés résistantes, et le Conseil consultatif colonial pour l'Agriculture et l'Hygiène animale a récemment recommandé d'envoyer dans le même but un botaniste au Brésil ou dans d'autres contrées où des

be found. Methods of control are being investigated on a severely infected property in Trinidad which has been acquired by the local Government for the purpose. The attempted control of the disease on estates by hand picking and destruction of the brooms is compulsory and is under the guidance and supervision of an inspecting staff, while further measures to reduce the losses by pruning and improved cultivation are being experimentally tested by the Agricultural Department on several estates. Quarantines against this disease are in force in the other British cacao-growing Colonies, and representations by the British Government* have advocated the imposition of similar quarantines in the French, Spanish, and Portuguese West African Colonies. This has been done in the French Colonies, but information regarding the Spanish and Portuguese is not available here.

III. — *Black root disease caused by several species of Rosellinia.*

This desease is caused in the West Indies by Rosellinia pepo and R. bunodes and is the most destructive disease of the crop in St. Lucia, while in the Gold Coast it is of minor importance and the species concerned there has not been identified. Like the next five diseases it is not confined to cacao, but attacks many tropical woody plants, and its control is now well understood. It is associated frequently with the use of certain susceptible shade trees, the replacement of which by resistant species (e. g. Gliricidia) is being pushed in St. Lucia. It is most prevalent in badly drained soils. Measures for its control by trenching to prevent spread, eradication of diseased plants, and soil aeration are discussed in W. Nowell's « Diseases of Crop Plants in the Lesser Antilles. » London West India Committee, 1923.

IV. — *Fomes root diseases, caused by Fomes lignosus (white root disease), F. noxius (brown root disease), and possibly other species.*

Brown root disease is the commonest root affection of cacao in the Gold Coast and New Guinea, and occurs also in Ceylon, Samoa, and elsewhere. White root disease is known in the Gold Coast, Sierra Leone, and probably Ceylon. They have been chiefly investigated as diseases of tea in Ceylon and India, and of rubber in Malaya, where fairly

* Cf.—Colonial Office Letter N° 70733/30 of 26-3-30 to the Foreign Office.

variétés résistantes pourraient être trouvées. Les méthodes de défense contre la maladie sont étudiées dans une propriété fortement infectée de La Trinité, qui a été acquise dans ce but par le Gouvernement local. On essaye de lutter contre la maladie dans certaines propriétés en rendant obligatoire le cueillage à la main et la destruction des « balais » et en plaçant ces opérations sous la direction et le contrôle d'inspecteurs. D'autres mesures tendant à réduire les pertes par l'élagage et par l'amélioration des méthodes de culture sont expérimentées par le Département de l'Agriculture dans plusieurs propriétés. Des quarantaines contre la maladie sont imposées dans les autres colonies britanniques qui produisent du cacao et le Gouvernement britannique a demandé l'imposition de pareilles quarantaines dans les colonies françaises, espagnoles et portugaises de l'Afrique occidentale (*). Cette mesure a été prise dans les colonies françaises, mais on ne possède pas de renseignements semblables en ce qui concerne les colonies espagnoles et portugaises.

III. — *Maladie noire des racines provoquée par différentes espèces de Rosellinia.*

Cette maladie est provoquée aux Indes occidentales par le Rosellinia pepo et le R. bunodes; elle constitue la maladie la plus funeste à la récolte dans l'île de Sainte-Lucie, tandis qu'elle présente une importance moindre à la Côte-de-l'Or, où l'espèce de parasite provoquant la maladie n'a pas été identifiée. De même que les cinq maladies suivantes, celle qui nous occupe n'est pas limitée au cacao, mais elle attaque de nombreux arbres tropicaux; elle peut actuellement être combattue d'une façon efficace. La maladie se présente souvent en corrélation avec l'emploi d'arbres ombrageants facilement attaquables; le remplacement de ces derniers par des espèces résistantes (par exemple, le Gliricidia) est actuellement poursuivi d'une manière intense dans l'île de Sainte-Lucie. La maladie fait surtout des ravages sur les terrains imparfaitement draînés. Les mesures de défense consistant dans le creusement de tranchées en vue d'éviter la propagation, dans le déracinement des arbres malades et dans l'aération du sol, sont exposées dans l'ouvrage de W. Novel : « Maladies des plantations dans les Petites-Antilles ». Londres, West India Committee, 1923.

IV. — *Maladies des racines provoquées par le Fomes lignosus (maladie blanche des racines), le Fomes noxius (maladie brune des racines) et probablement par d'autres espèces.*

La maladie brune des racines est l'affection radicale la plus commune du cacao à la Côte-de-l'Or et en Nouvelle-Guinée; elle apparaît également à Ceylan, dans les îles Samoa et ailleurs. La maladie blanche des racines est connue dans la Côte-de-l'Or, à Sierra-Leone et probablement à Ceylan. Ces maladies ont été étudiées principalement comme maladies

(*) Cfr. Lettre du Ministère des Colonies, n° 70733/30 du 26 mars 1930 adressée au Foreign Office.

satisfactory control measures have been worked out. Hitherto their incidence in cacao does not seem to have called for special measures to control them in the British Colonies, nor have any detailed investigations on them been carried out in respect of this crop by British mycologists.

V. — *Red root disease caused by Spaerostilbe repens.*

A minor disease recorded in the West Indies and Ceylon, where is it regarded as a sign of waterlogged soils and can usually be controlled by improved drainage. Another red root disease due to Ganoderma pseudaferreum is known in the East, but has not yet been reported in cacao in any British Colony.

VI. — *Collar crack caused by Armillaria melles.*

This is a major disease in the Gold Coast, and occurs also in Sierra Leone, and in the East African Colonies and Uganda, where it affects coffee, tea and other perennial crops. It was first reported on cacao by H. A. Dade in Togoland in 1922, and subsequently investigated by R. H. Bunting and H. A. Dade in the Gold Coast (Dept. of Agric. Bull. 5, 1927). The prescribed treatment in the Gold Coast, where it is a notifiable disease, consists in the removal and burning of all diseased material and the isolation of the affected area by trenches two feet deep.

VII. — *Collar rot caused by Ustulina zonata.*

This is a common affection of perennial woody crops in the tropics, but it is relatively infrequent in cacao, in which it is a minor disease in the Gold Coast and Ceylon. Extensive work in its control in rubber plantations has been carried on in Ceylon and Malaya, and these could be applied to cacao should it ever become necessary to treat the disease in this crop.

VIII. — *Pink disease due to Corticium salmonicolor.*

This, like the last five diseases, is widespread on perennial woody crops in the tropics and has been extensively investigated on Hevea rubber in Malaya. No serious outbreak on cacao has, as yet, been reported, but scattered cases occur in the West Indies, Ceylon, and Sierra Leone. Sufficient is known regarding its control to enable effective treatment on cacao if required.

du thé à Ceylan et aux Indes, et comme maladies du caoutchouc en Malaisie; dans ces différents pays, des mesures de défense efficaces ont pu être prises. Jusqu'à présent, leur apparition dans les plantations de cacao ne semble pas avoir nécessité de mesures de défense spéciales dans les colonies britanniques et les mycologistes britanniques n'ont pas procédé à des études détaillées de ces maladies par rapport aux plantations en question.

V. — *Maladie rouge des racines provoquée par le Spaerostilbe repens.*

La présence de cette maladie bénigne a été constatée aux Indes occidentales et à Ceylan, où elle est considérée comme l'indice de terrains trop humides; elle peut être ordinairement combattue en améliorant le drainage. Une autre maladie rouge des racines due au Ganoderma pseudaferreum est connue en Orient, mais son apparition dans les plantations de cacao des colonies britanniques n'a pas encore été signalée.

VI. — « *Collar crack* » *provoqué par l'Armillaria melles.*

Cette maladie constitue une affection sérieuse à la Côte-de-l'Or et se rencontre également à Sierra-Leone, dans les colonies de l'Afrique orientale et dans l'Uganda, où elle s'attaque au café, au thé et à d'autres cultures de plantes vivaces. Elle fut observée en premier lieu sur le cacao par H. A. Dade, dans le Togoland, en 1922, et a été étudiée ensuite par R.H. Bunting et H. A. Dade à la Côte-de-l'Or (Dép. de l'Agric., Bulletin 5, 1927). Le traitement prescrit à la Côte-de-l'Or, où les cas de maladie doivent être déclarés, consiste à enlever et à brûler tous les éléments malades et à isoler la zone infectée au moyen de tranchées d'une profondeur de deux pieds.

VII. — « *Collar rot* » *provoqué par l'Ustulina zonata.*

Cette maladie constitue une affection commune des cultures de plantes ligneuses vivaces dans les tropiques, mais elle est relativement rare en ce qui concerne le cacao, dont elle est une maladie bénigne à la Côte-de-l'Or et à Ceylan. Des études étendues concernant les moyens de défense contre cette maladie dans les plantations de caoutchouc ont été poursuivies à Ceylan et en Malaisie et ces moyens de défense pourraient éventuellement être appliqués aux plantations de cacao.

VIII. — *Maladie rose due au Corticium salmonicolor.*

Cette maladie, comme les cinq dernières, est largement répandue dans les cultures de plantes ligneuses vivaces des Tropiques et a été étudiée d'une façon approfondie sur l'arbre à caoutchouc Hevéa en Malaisie. Jusqu'à présent, cette maladie ne paraît pas avoir atteint sérieusement le cacao, mais des cas isolés se sont présentés aux Indes occidentales, à Ceylan et à Sierra-Leone. On connaît suffisamment les moyens de défense contre la maladie pour permettre, le cas échéant, un traitement efficace en ce qui concerne les plantations de cacao.

IX. — *Die-back and brown pod rot caused by Botryodiplodia theobromae.*

Usually regarded as a major disease of cacao in practically all cacao-growing countries, this fungus is said to be chiefly destructive on the pods, having been reported to be responsible for heavy losses of these in the West Indies and elsewhere. In Uganda its attacks on the twigs are reported to cause extensive die-back, but Nowell has pointed out that in Trinidad this condition is mainly due to neglected cultivation, the fungus being only weakly parasitic on the twigs. This investigator is also inclined to think that much of the pod damage ascribed to brown pod rot is really consequent on an earlier attack of Phytophthora, and in careful studies on pod diseases in the Gold Coast, H. A. Dade found B. theobromae was responsible for only 0.3 per cent. as against 40.5 per cent. due to Phytophthora, out of a total of 55.4 per cent. rotting of the crop in certain plots. It seems probable, therefore, that this disease is a symptom of some underlying primary trouble and that direct treatment is not required. The same considerations probably apply to another form of die-back, associated with Glomerella cingulata, that has been reported in Uganda, and while the same or a closely allied fungus caused 6.2 per cent. of the pod rotting studies in the Gold Coast and also attacks the pods in Trinidad and Ceylon, it is not regarded as of economic importance.

X. — *Mealy pod disease due to Trachysphaera fructigena.*

Discovered in the Gold Coast in 1919, this fungus appears to be confined to West Africa where it was investigated chiefly by R. H. Bunting in the Gold Coast. It penetrates most readily into injured fruit when young, but can also rot the pods after picking. Its biology and control form part of the extended study of pod diseases on which H. A. Dade is engaged (cf. Gold Coast Dept. of Agric. Bull. 23, pp. 109-128, 1931).

XI. — *Minor diseases of the twigs and leaves.*

The thread blights (white, due to Marasmius scandens; brown, due to M. byssicola; and purple-brown, probably due to another species of the same genus) are fairly common in West Africa but are easily controlled by cutting out. In the West Indies a similar disease is caused by Corticium koleroga. Red rust due to Cephaleuros parasitica

IX. — *Dépérissement (Die-back) et maladie brune des cabosses, provoquée par la Botryodiplodia theobromae.*

Cette maladie, qui est ordinairement considérée comme une affection sérieuse du cacao dans presque tous les pays producteurs, est provoquée par un champignon qui exerce surtout ses ravages sur les cabosses. Il a été rendu responsable de lourdes pertes aux Indes Occidentales et ailleurs. Dans l'Uganda, il attaque les rameaux et provoque un dépérissement accentué; toutefois, Nowell a fait remarquer qu'à La Trinité ce phénomène est principalement dû à une culture négligée, étant donné que le champignon en question constitue un parasite très peu résistant sur les rameaux. Ce chercheur est également enclin à croire que les dégâts aux cabosses attribués à la maladie brune des cabosses sont en réalité la conséquence d'une attaque antérieure par le Phytophthora. A la suite d'études sérieuses sur les maladies des cabosses à la Côte-de-l'Or, H. A. Dade a établi que les dégâts aux récoltes dans certaines plantations, s'élevant à un total de 55.4 p. c., doivent être attribués pour 0.3 p. c. seulement au B. theobromae, mais, par contre, pour 40.5 p. c. au Phytophthora. En conséquence, il semble probable que la maladie qui nous occupe ne soit que le symptôme d'une affection primaire sous-jacente et qu'un traitement direct ne soit pas nécessaire. Les mêmes considérations s'appliquent probablement à une autre forme de dépérissement provoqué par le Glomerella cingulata, qui a été signalé dans l'Uganda, et bien que le même champignon ou un autre d'une espèce très voisine ait causé 6.2 p. c. des cas étudiés de dépérissement des cabosses à la Côte-de-l'Or et attaque également les cabosses à La Trinité et à Ceylan, cette maladie n'est pas considérée comme ayant une grande importance au point de vue économique.

X. — *Maladie poudreuse des cabosses (Mealy pod disease) provoquée par le Trachysphaera fructigena.*

Découvert à la Côte-de-l'Or en 1919, ce champignon paraît être confiné à l'Afrique occidentale, où il a été étudié principalement par R. H. Bunting, à la Côte-de-l'Or. Il pénètre très promptement dans les fruits blessés lorsqu'ils sont jeunes, mais il peut également provoquer le dépérissement des cabosses après le cueillage. L'étude biologique de ce champignon, ainsi que celle des moyens de défense contre ses attaques font partie de l'étude approfondie des maladies des cabosses entreprise par H. A. Dade (cfr. Depart. de l'Agric. de la Côte-de-l'Or, Bulletin 23, pages 109 à 128, 1931).

XI. — *Maladies bénignes des rameaux et des feuilles.*

La maladie filiforme (blanche, provoquée par le Marasmius scandens; brune, provoquée par le M. byssicola; et pourpre-brune, probablement due à une autre espèce du même genre) est très commune en Afrique occidentale, mais peut être facilement combattue en coupant les parties malades. Aux Indes Occidentales, une maladie similaire est occa-

and C. minimus occurs chiefly on the twigs in Trinidad, the Gold Coast, Uganda and Ceylon, but is known in most parts of the tropics. It has not yet been found to be of economic importance on cacao, except occasionally in Trinidad. None of the leaf diseases recorded on this crop appears to require treatment.

XII. — *Mould damage to cacao beans in fermenting and transit.*

This subject has been investigated chiefly by Dade in the Gold Coast (Dept. of Agric. Bull. 16, pp. 74-100, 1929), Laycock in Nigeria (9th Ann. Bull. Agric. Dept. pp. 5-26, 1930), and F. R. Passmore and R. G. Bunting at the Stored Products Laboratory, Slough (cf. Gold Coast Dept. of Agric. Bull. 16, pp. 37-57, 1929, and Bull. Impl. Inst., 3, pp. 296-305, 1932). These studies have shown how mould trouble may be avoided where the moisture content can be controlled.

Certain diseases of cacao not yet reported in any British Colony have been recorded in Ecuador, the Dominican Republic, and the island of St. Thomas in the Gulf of Guinea. Of these the most serious appear to be the Monilia and Sphaeronema diseases in Ecuador. Little recent work on these appears to have been done.

The British mycologists engaged in the investigation of cacao diseases in the Gold Coast and Trinidad, where alone serious work is being attempted, have many other duties as in each case they are charged with the supervision of the health of all the cultures in the Colony. There is no whole-time officer engaged in the general study of the cocoa tree in a diseased state. Should any new disease appear it would have to be attacked as an ad hoc problem.

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sionnée par le Corticium koleroga. La rouille rouge, due au Cephaleuros parasitica et au C. minimus, apparaît surtout sur les rameaux à La Trinité, à la Côte-de-l'Or, dans l'Uganda et à Ceylan, mais elle est connue dans la plupart des régions des Tropiques. Il n'a pas encore été établi que sa présence sur les plantations ait une importance économique, si ce n'est occasionnellement à La Trinité. Aucune des maladies des feuilles atteignant la culture du cacao ne semble nécessiter un traitement spécial.

XII. — *Dégâts causés par la moisissure aux fèves de cacao par suite de fermentation ou en cours de transport.*

Cette question a été principalement étudiée par Dade à la Côte-de-l'Or (Départ. de l'Agric., Bull. 16, pages 74-100, 1929), par Laycock au Nigéria (9e bull. annuel du Départ. de l'Agric., pages 5-26, 1930) et par F. R. Passmore et R. G. Bunting au Laboratoire des produits emmagasinés à Slough (cfr. Départ. de l'Agric. de la Côte-de-l'Or, Bull. 16, pages 37-57, 1929, et le Bull. Impl. Inst. 3, pages 296-305, 1932). Ces études ont montré qu'il est possible d'éviter la moisissure en contrôlant la teneur d'humidité.

Certaines maladies encore inconnues dans les colonies britanniques, ont été signalées dans l'Equateur, la République Dominicaine et l'île de San-Thomé dans le golfe de Guinée. Parmi celles-ci, les plus sérieuses paraissent être les maladies « Monilia » et « Sphaeronema » dans l'Equateur. Il semble qu'on ait fait très peu d'études récentes concernant ces maladies.

Les mycologistes britanniques qui s'occupent de l'étude des maladies du cacao à la Côte-de-l'Or et à La Trinité, seuls pays où on se soit efforcé de faire du travail sérieux, sont encore astreints à d'autres besognes, vu qu'ils sont chargés d'une façon constante du contrôle sanitaire de toutes les cultures dans la colonie. Aucun agent ne peut consacrer tout son temps à l'étude générale des maladies du cacaotier. Si une nouvelle maladie devait se manifester, le problème de la lutte contre cette maladie se poserait tout entier.

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Conférence Internationale du Cacao

(Propagande internationale pour les produits à base de cacao)

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Franco, contre envoi de **10 belgas à l'Office International des Fabricants de Chocolat et de Cacao**

IIIe Congrès International technique et chimique des Industries Agricoles.

Sur l'initiative du Gouvernement français et de l'Association des Chimistes de Sucrerie, de Distillerie et des Industries Agricoles de France et des Colonies, le IIIe Congrès International technique et chimique des Industries Agricoles se réunira à Paris, au *Conservatoire National des Arts et Métiers* et à l'*Ecole Centrale des Arts et Manufactures*, du 25 au 31 mars 1934, sous la présidence de M. Fernand DAVID, Sénateur, ancien Ministre, Membre de l'Académie Nationale d'Agriculture de France.

Le programme technique du Congrès est l'œuvre d'une Commission Internationale qui comprend les représentants qualifiés des trente-trois Gouvernements étrangers ayant, à ce jour, officiellement donné leur adhésion au Congrès.

Cette Commission a, tout d'abord, arrêté le Règlement de celui-ci et réparti les travaux en groupes et sections.

En ce qui concerne les questions intéressant notre industrie, signalons que le Groupe II traitera des industries sucrières et que le Groupe IV est consacré aux industries alimentaires : chocolaterie, matières grasses, meunerie, laiterie, etc...

La section « Chocolaterie » étudiera les questions ci-dessous, qui sont toutes d'un très grand intérêt pour notre industrie :

1° Standardisation des méthodes officielles d'analyse des cacaos en poudre, des beurres de cacao et des chocolats.

Rapporteur : M. le Professeur Vivario, de l'Université de Liége;

2° Evolution et nouvelles données concernant la fabrication et la consommation du chocolat.

Rapporteur : M. le Professeur Henri Labbé, de la Faculté de Médecine de Paris;

3° Utilisation de la lécithine dans la fabrication du chocolat.

Rapporteur : Mme Lefèvre, Assistante à la Faculté de Médecine de Paris;

4° Recherche des beurres factices dans la composition du chocolat.

Rapporteurs : M. Kling, Directeur du Laboratoire municipal à la Préfecture de Police;

M. R. Lerat, Préparateur à la Faculté de Médecine de Paris;

5° Lutte contre la mite du cacao.

Rapporteur : M. Vayssière, professeur de Zoologie agricole à l'Institut d'Agronomie coloniale à Nogent-s/Marne.

Puis, persuadée que, seule l'étude approfondie d'un nombre restreint de questions est susceptible de servir utilement à l'avancement de la science et de l'industrie, la Commission Internationale a déterminé quels seraient les sujets d'ordre général sur lesquels seraient présentés au Congrès, des rapports qu'elle a confiés à des personnalités internationales hautement qualifiées, en même temps qu'elle demandait à d'autres des communications restreintes à l'étude d'un point particulier de ces questions.

Parmi les sujets généraux qui seront ainsi étudiés, il convient de citer :

La détermination du pH et du rH;

L'amélioration de la betterave par la sélection génétique;

L'épuration des jus sucrés;

La cristallisation et la conservation des sucres;

Les qualités exigées pour le pain et les propriétés correspondantes des farines et des blés;

L'emploi des huiles végétales dans les moteurs;

L'évolution de la fabrication du chocolat.

En annexe au Congrès sera organisée une Exposition Internationale d'appareils industriels et instruments de laboratoire, dont l'organisation a été confiée à M. Robert GALLOIS, Ingénieur-Chimiste, secrétaire général adjoint du Congrès.

Le Congrès sera solennellement ouvert par M. le Président de la République, assisté des Membres du Gouvernement et en présence des Membres du Corps Diplomatique, le lundi 26 mars, à 10 heures du matin. La clôture en sera prononcée le samedi 31 mars.

Les Congressistes seront, au cours de cette semaine, l'objet de nombreuses réceptions, notamment par M. le Président de la République, le Ministre des Affaires Etrangères, le Président du Conseil Municipal, le Président de la Chambre de Commerce de Paris.

Des promenades sont également organisées pour les familles des Congressistes, dans les Musées de Paris, à l'Hôtel des Invalides, dans les Châteaux de l'Ile-de-France, aux Manufactures Nationales de Sèvres et des Gobelins, dans les Maisons de Couture, etc...

Enfin, à l'issue du Congrès, un voyage d'études aura lieu à travers les principales régions viticoles et brassicoles de France, avec le concours le plus empressé des autorités locales.

Pour tous renseignements, il convient de s'adresser à M. Henry-François DUPONT, Avocat-Conseil du Ministère de l'Agriculture, Vice-Président de l'Association des Chimistes, Secrétaire Général du Congrès, 156, boulevard de Magenta, Paris (Téléphone : Trudaine : 26-56. Adresse télégraphique : Chimicongrès Paris).